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PHYTOPATHOLOGY

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AUGUST, 1933

TWO LEAF SPOTS OF THE PEANUT (ARACHIS HYPOGAEA L.)[1]

NAOMI C. WOODROOF

(Accepted for publication August 26, 1932)

The peanut is an extensively grown and valuable crop in many parts of the world. Large acreages are grown in the southeastern United States, South Africa, parts of South America, the West Indies, Philippine Islands, Japan, India, Australia, Java, Italy, and in China. Seed from these widely separated regions have been exchanged, and as a consequence it is probable that diseases have become widely disseminated throughout most of these areas. The prevalence of peanut leaf spots is shown by the fact that they are mentioned in literature from all of these countries.

In 1928, during the course of some preliminary seed treatment tests, it was noted that there were 2 distinct leaf spots occurring in abundance on the leaves of peanut plants growing in the field at the Georgia Experiment Station. Further studies were begun to determine which of these spots was originally described by Berkeley (4) as *Cladosporium personatum* and if this was the same as that studied by Ellis and Everhart (10) when they changed the name of the causal organism to *Cercospora personata*. Fungi other than *C. personata* have been described as causing spotting of the leaves of the peanut plant, but most of these have been considered synonymous by subsequent writers. The present investigation is an attempt to clarify the resultant confusion by comparing the various descriptions and herbarium specimens and by comparing these with the 2 spots found in Georgia.

LITERATURE REVIEW

In the following literature review only those references are listed that give sufficient detail to enable the reader to determine rather accurately the systematic classification of the disease described. The writers who include in their papers a synonymy of the respective fungi are listed in table 1.

In 1875 Berkeley (4, p. 106) published the first description of a peanut leaf spot and gave the name *Cladosporium personatum* to the causal fungus. This was the result of a study of material collected by Ravenel in South Carolina in 1855.

[1] Published with the approval of Director as Paper No. 36—Journal series Georgia Experiment Station.

Ten years later Ellis and Everhart (10), after examining material collected in Alabama and South Carolina, transferred the fungus to the genus Cercospora. This change has been generally accepted and the name *Cercospora personata* used exclusively in preference to *Cladosporium personatum*.

In 1891 Atkinson (1, p. 43) examined material collected in Alabama and South Carolina that was reported to agree with specimen N. A. F. No. 2,480 collected by Professor Tracy and determined as *Cercospora personata* by Ellis and Everhart. Atkinson, however, reports a greater conidial length and more septations than are recorded by Ellis and Everhart. It is possible that Atkinson examined leaves infected by 2 different fungi, as he found the conidiophores to be amphigenous, whereas Ellis and Everhart describe the fungus as being hypophyllous.

In Raciborski's (18) description of *Septogloeum arachidis* appear 2 statements that become significant when compared with the illustrations and descriptions of later writers. These are, first, "*Das Mycelium des Pilzes breitet sich am stärksten zwischen den Schwammparenchymzellen aus und sendet in diese charakteristische gelappte Haustorien,*" and, second, "*Flecken konzentrisch gruppirte.*"

Hennings (12) described *Cercospora arachidis* from a study of material collected in Brazil and says he classifies it as a new species, because "*Von C. personata* (B. & C.) Ell. *durch die aufgeblasenen Flecke, die septirten Hyphen, die kürzeren, 3–6 septirten conidien der Beschreibung nach verschieden.*" It seems possible Ellis and Everhart failed to mention the fact that their fungus produced raised spots or that they covered this point in a statement regarding the densely tufted conidiophores. They also probably failed to observe that the conidiophores were septate in some instances. The variation in the conidial length does not seem great enough to be important. Later writers have commonly listed *C. arachidis* as synonymous with *C. personata*.

Zimmermann (29, Taf. 2, Fig. 9) illustrated the conidiophores and the conidia of *Septogloeum arachidis*. From these drawings and the accompanying description it was concluded that Zimmermann studied the same leaf spot as described by Raciborski. A comparison of Raciborski's collection with N. A. F. No. 2,480 shows that *S. arachidis* is the same as *Cercospora personata*.

Bancroft (3, pp. 52 and 55) reports both *Cercospora personata* and *Septogloeum arachidis* from the West Indies. The more complete description of *S. arachidis* may indicate that he was more familiar with descriptions of this leaf spot. His description of *C. personata* is similar to that of Ellis and Everhart. Possibly he knew of a second leaf spot that he believed described by these writers as *C. personata*.

TABLE 1.—*The writers who give or suggest synonymous relationships of the peanut leaf-spotting fungi are listed chronologically as follows:*

Authority	Date	Reference no. in cited literature	SYNONYMS (The first name given is that accepted by each writer)
Ellis and Everhart	1885	10	*Cercospora personata* (B. & C.) E. & E. *Cladosporium personatum* B. & C.
Chevalier	1913	7, p. 72–73	*Septogloeum arachidis* Rac. *Cercospora personata* (B. & C.) E. & E. *C. arachidis* P. Henn.
Wolf	1914	27	*Cercospora personata* (B. & C.) E. & E. *Cladosporium personatum* B. & C. *Cercospora arachidis* P. Henn.
Yates	1917	28, p. 315	*Cercospora personata* (B. & C.) E. & E. *Cladosporium personatum* B. & C. possibly *Septogloeum arachidis* Rac.
Van Der Bijl	1920	5	*Septogloeum arachidis* Rac. *Cercospora personata* (B. & C.) E. & E.
Wakefield	1921	25, p. 299	*Cercospora personata* (B. & C.) Ell. *Septogloeum arachidis* Rac.
Sawada	1922	22	*Cercospora personata* (B. & C.) E. & E. *C. arachidis* P. Henn. *Septogloeum arachidis* Rac.
Maublanc	1924	17	*Cercospora personata* (B. & C.) E. & E. *Cladosporium personatum* B. & C. *Septogloeum arachidis* Rac. *Cercospora arachidis* P. Henn.
Mason	1928	16	*Cercospora personata* (B. & C.) E. & E. *Septogloeum arachidis* Rac.
Curzi	1931	9	*Cercospora personata* (B. & C.) E. & E. *Cladosporium personatum* B. & C. *Septogloeum arachidis* Rac. *Cercospora arachidis* P. Henn.
Solheim and Stevens	1931	24, p. 368–369	*Cercospora personata* (B. & C.) E. & E. *Cladosporium personatum* B. & C. *Cercospora arachidis* P. Henn.
Saccardo	1886	21	*C. personata* (B. & C.) E. & E. *Cladosporium personatum* B. & C.

Heald and Wolf (11, p. 49) may have studied a mixed infection, but it is quite possible that they collected only specimens infected with the Cercospora producing long slender obclavate conidia that they illustrate as *C. personata*. They state that the conidiophores are amphigenous though more appear on the lower surface.

Butler's (6) description and illustrations of *Septogloeum arachidis* are similar to those of Zimmermann. They were both undoubtedly dealing with the same type leaf spot. Butler is the first writer to illustrate haustoria in the cells of the leaf tissue. This writer states that *Cercospora personata* also occurs in one province in India but did little damage.

Wolf (27) undoubtedly has described a mixed infection as *Cercospora personata*. His illustrations show slender obclavate conidia and 2 types of conidiophore tufts, 1 small and having a less definitely stromatic base, while those of the second are large and stromatic. The former was produced epiphyllously and the latter hypophyllously. The statement is made that the large definitely stromatic conidiophore tufts are not typical of the genus Cercospora. This writer considered *Septogloeum arachidis* as described by Butler from India to be a separate leaf disease of the peanut, though he was aware that Chevalier (7) had considered it identical with *C. personata*. Wolf considered *C. arachidis* synonymous with *C. personata*.

Shaw (23) and Mackenna (14, p. 85) reported in 1917–18 2 leaf spots of the peanut in India, 1 *Cercospora personata* and the other an unknown species of Cercospora. Neither writer described these 2 fungi.

Welles (26), in 1917, illustrated *Cercospora personata* from the Philippine Islands as having long, slender, obclavate conidia. A year later Reinking (19, pp. 176–177) described and illustrated the fungus of a leaf spot of peanuts that had short, thick, abruptly obclavate or cylindrical conidia and that was considered to be *Septogloeum arachidis*. In 1919 this same writer (20, pp. 114–115) referred to *C. personata* but stated that, "Further study will probably show that the Philippine leaf spot is also a species of Septogloeum." It is probable that he referred to the same fungus in both cases.

In 1917 Hori[2] (13, p. 26) described from Japan a Cercospora leaf spot of the peanut to which he gave the name *C. arachidicola*. The original description by Hori has not been read, but a comparison of a specimen collected by Hori, with the descriptions and illustrations by Sawada (22),[3] has enabled the writer to determine that *C. arachidicola* is the same as the long, slender-spore Cercospora commonly found on peanuts in southeastern United States that other writers have often confused with *C. personata*.

[2] Since this paper was submitted for publication a letter was received from M. Ishikawa inclosing a translation of Hori's description of *Cercospora arachidicola*, and also one of Sawada's paper. The translation of the former is particularly valuable because the writer has been unable to obtain a copy of the original in America. Hori's description is very brief, and his principal claim for priority lies in the type specimen which he prepared.

[3] The writer is very grateful to Mr. Geo. T. Akamatsu for an English translation of this reference.

Sawada's (22) illustrations of *Cercospora personata* are similar to those of Zimmermann, Butler, and Reinking, for *Septogloeum arachidis,* and of Maublanc and Bal for *C. personata.* His illustrations of *C. arachidicola* are similar to those of Welles, Heald and Wolf, and Wolf.

Maffei (15), in 1921, described a Cercospora with long, slender spores on peanut leaves collected in Italy, which he classified as a variety of *Cercospora arachidis* (*C. arachidis* var. *macrospora*). This writer considered *C. arachidis* as distinct from *C. personata.* No reference is made to Hori's description of *C. arachidicola.*

Only Hori and Maffei have actually described this species of Cercospora on the peanut; and, as Hori's description has priority, *C. arachidicola* must be accepted as the name of this fungus rather than Maffei's *C. arachidis* var. *macrospora.*

Bal (2), 1921, was the first writer to illustrate the fungus having thick, abruptly obclavate conidia under the name *Cercospora personata.*

In 1924 Maublanc (17) published drawings of *Cercospora personata* that are very like those of Zimmermann, Butler, Bal, Reinking, and Sawada, for *C. personata* and *Septogloeum arachidis.* Maublanc also expresses the opinion that this fungus possibly should not be classified as a Cercospora. *Cercospora personata* is considered as synonymous with *S. arachidis* and *C. arachidis;* whereas, it is stated that *C. arachidis* var. *macrospora* may possibly be a different species.

Miss Wakefield (25, p. 299) in 1921, and Mason (16) in 1928, list *Septogloeum arachidis* as a synonym of *Cercospora personata.*

Ciferri (8), in 1925, expressed the opinion that possibly there was a Cercospora other than *C. personata* attacking peanuts in Dominica.

Solheim and Stevens (24, pp. 368–369) include much detail in their description of *Cercospora personata,* which is evidently the same fungus as that of Ellis and Everhart. *Cladosporium personatum* and *C. arachidis* are given as synonyms, but no mention is made of *Septogloeum arachidis, C. arachidis* var. *macrospora,* or *C. arachidicola.*

Curzi (9) considers *Cercospora arachidis* and *Septogloeum arachidis* as synonymous with *C. personata* but expresses a doubt regarding *C. arachidis* var. *macrospora* in the statement "*Essa e stata descritta concaratteri corrispondenti pui a una specie distincta che a una varieta.*"

Saccardo (21) lists *Cercospora personata* and *Septogloeum arachidis* and cites *Cladosporium personatum* as a synonym of the former. No reference is made to *Cercospora arachidicola* and *C. arachidis* nor to the variety of the latter described by Maffei.

MATERIALS AND METHODS

Material for study included leaves from field-grown as well as leaves from artificially inoculated plants grown in the greenhouse. Material for

microscopical examination was fixed in Flemming's weaker solution or in a solution of picric and sulphurous acids in 75 per cent alcohol. Microtome sections were cut 6 to 8 μ in thickness and stained with Heidenhain's Iron-alum Haematoxylin.

In studying some of the herbarium specimens blocks of the leaves were soaked in a solution of potassium hydroxide for 24 hours, thoroughly washed, and fixed in Flemming's weaker solution.

Artificial inoculations were successfully made in a greenhouse with plants kept in a moist chamber. These chambers were frames covered with water-proof Cellophane or with Cell-o-glass. It was necessary to keep the plants covered in the greenhouse in order to obtain abundant infection and sporulation of the fungus.

A COMPARISON OF HERBARIUM SPECIMENS OF LEAF SPOTS OF THE PEANUT

The examination of herbarium specimens aided greatly in clarifying the last doubtful points regarding the nomenclature of the 2 fungi attacking the leaves of *Arachis hypogaea* L.

The following specimens have been examined:[4]

Ravenel, Fungi Caroliniani, Fasc. III, 1855.

85. *Cladosporium personatum* Berk. et Curt. foliis Arachidis hypogeae. Ex. Alabama quoque misit Beaumont foliis Cassiae occidentalis.

Ellis and Everhart, North American Fungi, Second Series.

2480. *Cercospora personata* (B. & C.) E. & E. Starkville, Mississippi, Oct. 1888. Prof. Tracy.

M. Raciborski, Cryptogammae Parisiticae, Insula Java Lectae.

Nr. 50. *Septogloeum arachidis* Rac. In foliis Arachi hypogaei. Tegal Leg. Raciborski.

Cercospora arachidicola Hori.

Collected by S. Hori Sept. 19, 1918. Matsudo, Prov. Sehimosa.

Cercospora personata (B. & C.) E. & E.

J. C. Hopkins, Southern Rhodesia M. R. 1619. Amani, Tanganyika.

Cercospora personata (B. & C.) G. B. Wallace, Tanganyika. No. 1161.

Cercospora personata (B. & C.) E. & E. F. Eyles, Southern Rhodesia. Nos. 4987 and 5001.

Cercospora personata (B. & C.) E. & E. G. H. Jones, Nigeria (West Africa).

Microtome sections of the first 3 specimens were prepared and examined. Ravenel's collection described by Berkeley was found on examination to

[4] The writer is grateful to the following for the opportunity to examine herbarium specimens: Dr. C. L. Shear, Bureau of Plant Industry, Washington, D. C., and to Dr. S. F. Ashby, Imperial Mycological Institute, Kew, Surrey, England.

have conidiophores developing on definite stromata, produced hypophyllously. There was much intercellular mycelium sending many branched haustoria into the cells of the spongy mesophyll and palisade tissues. No conidia were observed either in sections or in mounts from the spots. However, an examination of a specimen of a similar leaf spot (*Cladosporium personatum* B. & C. var. *occidentalis*) on *Cassia occidentalis* showed many short obclavate or cylindrical conidia. This point, together with the internal mycelium, haustoria, and hypophyllous conidiophores of Ravenel's type specimen on peanut leaves, shows that Berkeley examined the same leaf spot as that studied later by Raciborski, Zimmermann, Ellis and Everhart, *et al.*

The collection (N. A. F. No. 2,480), determined by Ellis and Everhart as *Cercospora personata*, and Raciborski's specimen of *Septogloeum arachidis* were both found to be the same as Ravenel's collection described by Berkeley. The large stromatic conidiophore clusters and the haustoria are the same in all 3 specimens. (Figs. 1, G; 2, A).

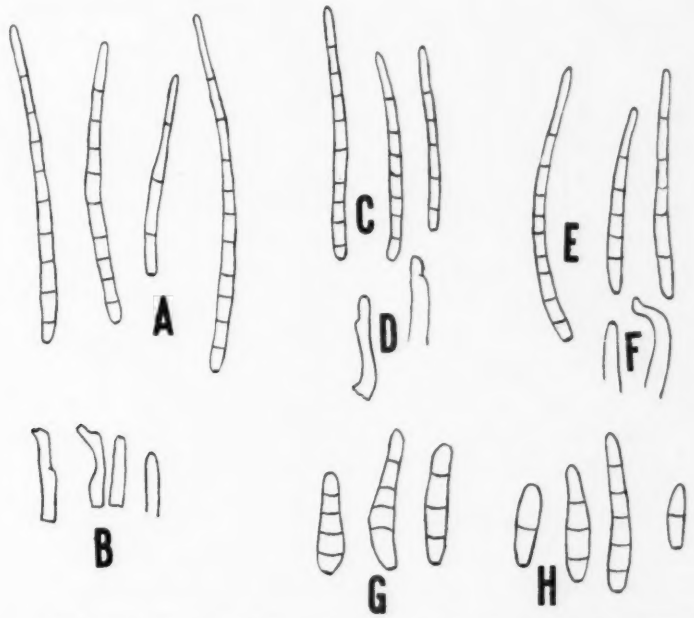

FIG. 1. Conidia and conidiophores. ×540. A–B. *Cercospora arachidicola* from fresh material collected in Georgia. C–D. *C. arachidicola*, type specimen collected by Hori in Japan. E–F. *C. arachidicola* collected by Eyles in South Rhodesia. G. *C. personata* N. A. F. No. 2480 identified by Ellis and Everhart. H. *C. personata* from material collected in Nigeria.

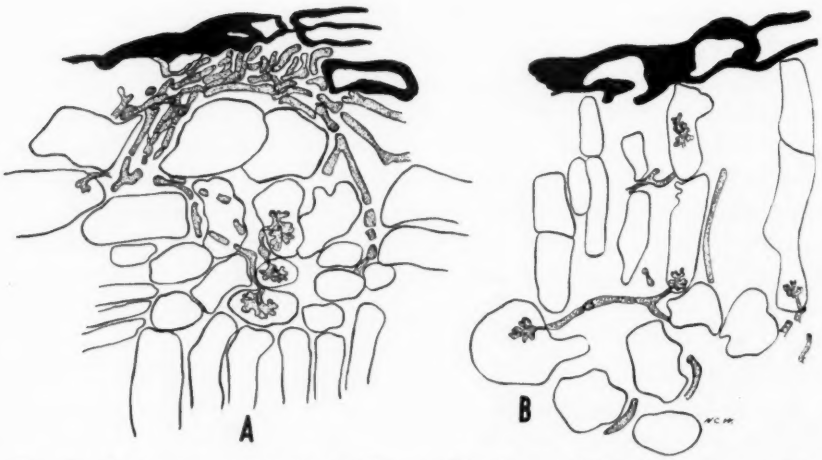

FIG. 2. *Cercospora personata.* ×493. A. Mycelium and haustoria from specimen classified as *Septogloeum arachidis* by Raciborski. B. Haustoria and internal mycelium from material collected in Georgia.

Hori's specimen of *Cercospora arachidicola* was found to have long, slender, obclavate conidia. The halos about the spots are distinct (Fig. 1, C–D).

The specimens of *Cercospora personata* collected by J. C. Hopkins in South Rhodesia were found to have long, slender, obclavate conidia. The same is true of the collections by Eyles from the vicinity of Salisbury in South Rhodesia (Fig. 1, E–F). The specimens collected by Wallace in Tanganyika and those of Jones in Nigeria were found to have short, thick, abruptly obclavate or cylindrical conidia. (Fig. 1, H). The conidiophore tufts on the lower surface of the leaf are concentrically arranged in many of the spots. The 2 latter are the same fungus as the type specimens of *C. personata, Cladosporium personatum,* and *Septogloeum arachidis;* whereas, the first 3 are the same as the species described by Hori as *Cercospora arachidicola* and probably by Maffei as *C. arachidis* var. *macrospora.* They are also the same as a leaf spot found in Georgia.

COMPARATIVE DESCRIPTIONS OF TWO PEANUT LEAF SPOTS AND THEIR RESPECTIVE CAUSAL FUNGI

Both microscopically and macroscopically the 2 leaf spots attacking peanut leaves are quite easily distinguished, especially when the spots become older.

Cercospora arachidicola hori. Syn. *C. arachidis* P. Henn. var. *macrospora* Maff.

Cercospora arachidicola produces irregularly circular, often confluent spots varying in size from 1 mm. to 1 cm. in diameter and surrounded by

a bright yellow halo, blending into the green of the leaf. Initial spots are yellowish specks. The color of the older spots varies from dark brown to almost black on the upper surface of the leaf but is usually a lighter brown color on the under surface. The yellow border is less pronounced on the lower than on the upper surface. (Fig. 3, A). Elliptical spots are often found on the stems and leaf petioles later in the season.

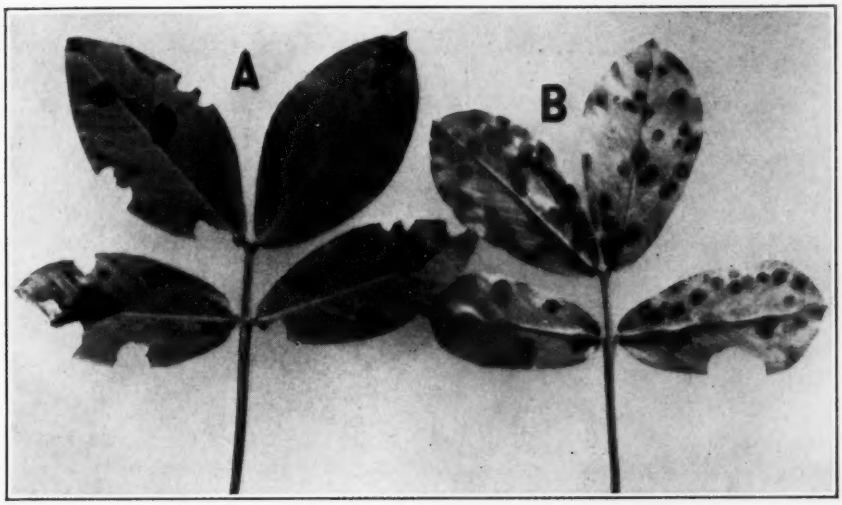

FIG. 3. A. Upper surface of peanut leaf showing large irregular spots of *Cercospora arachidicola*. B. Lower surface of peanut leaf showing the raised round spots of *C. personata*.

The mycelium is both internal and external. No haustoria have been noted. The mycelium is both intracellular and intercellular.

The conidiophores of *Cercospora arachidicola* are amphigenous. In young spots the conidiophores are produced almost exclusively on the upper surface of the leaf, but in older spots a few may sometimes be found on the lower surface. The conidiophores are produced in 3 different ways; first, they emerge directly through a stoma, forming a small fascicle of conidiophores; second, the mycelium grows out between epidermal cells or through a stoma and continues to develop under the cuticle, and from this mycelium fascicles of conidiophores are formed; third, conidiophores form from mycelium growing in epidermal cells that are later ruptured by the developing conidiophores. The bases of the conidiophore groups are usually somewhat stromatic, especially when mature. (Fig. 4).

The conidiophores are subgeniculate $21.6–40.5 \times 3.2–5.4\ \mu$, yellowish brown, continuous or 1- to 2-septate. The scars marking the point of at-

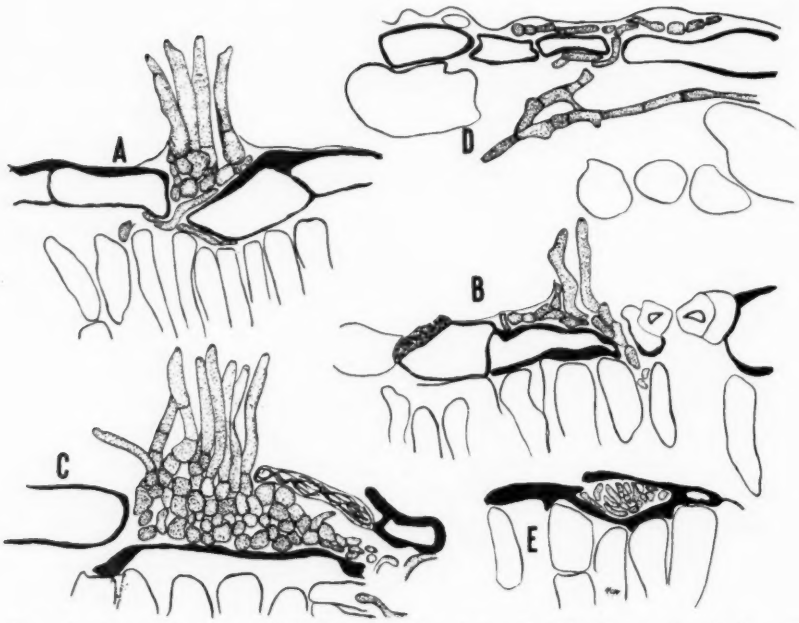

FIG. 4. *Cercospora arachidicola.* ×493. A–B. Conidiophores developing from mycelium beneath the cuticle on the upper surface of a leaf. C. Mature group of conidiophores showing the stromatic base. D. Mycelium growing beneath the cuticle on the lower surface of the leaf. E. Mycelium of a young conidiophore fascicle developing within an epidermal cell.

tachment of the conidia are plainly visible. The colorless, or pale yellowish to slightly olivaceous, obclavate conidia, measure 37.8–108 × 2.7–5.4 μ and are 4- to 12-septate, the usual number being 5 to 7. In dry weather the number of septations are fewer. (Fig. 1, A–B).

The conidia germinate in water and in slight accumulations of water on the surface of cover glasses placed over a van Tiegham cell. The manner in which they germinate depends on the amount of moisture present.

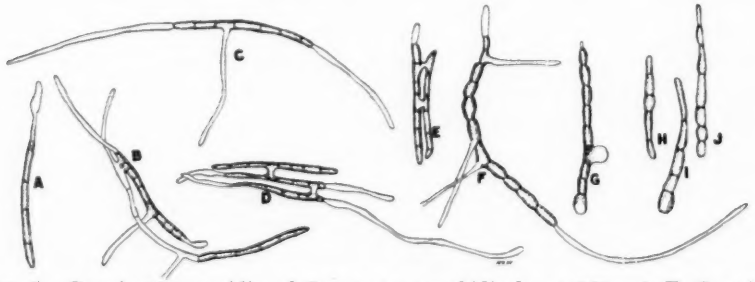

FIG. 5. Germinating conidia of *Cercospora arachidicola.* ×360. A–F. Germinated in heavy film of water on cover glass placed over water in a van Tiegham cell. G–J. Swelling of cells in a film of water too thin for ready germination.

Under favorable conditions long germ tubes develop in about 12 hours from any of the several cells of the conidium, often several tubes from a single conidium. With insufficient moisture the cells may swell without developing germ tubes (Fig. 5).

Spermogonia develop on fallen leaves late in September or early in October. At about the time the spermogonia begin to develop the internal mycelium becomes very thick walled.

In artificial culture the mycelium grows very slowly, forming a dense black mass. Conidia have not been observed in culture.

Cercospora personata (B. & C.) E. & E. Syn. *Cladosporium personatum* B. & C. *Septogloeum arachidis* Rac. *Cercospora arachidis* P. Henn.

Cercospora personata spots are circular and measure from 1 to 7 mm. in diameter. Halos are wanting or are a pale yellowish green on the lower surface. Bright yellow halos develop around the more mature spots on the upper surface of the leaf.

The mycelium is internal, no external mycelium having been observed. Branched haustoria develop in the palisade and spongy mesophyll cells (Fig. 2, B). Late in the season the mycelium becomes thick walled.

The conidiophores are amphigenous but more numerous on the lower surface, where the tufts are often arranged concentrically, especially in the larger spots. These can be seen easily with the naked eye. (Fig. 3, B). Elliptical spots often appear on the stems and on the leaf petioles.

The conidiophores measure 24.3–54 × 2.7–8.1 μ and are continuous, or 1- to 2-septate, subgeniculate or shouldered, the points of attachment of the conidia being plainly visible. The color is reddish brown and the apices are often hyaline. The conidiophores develop from loosely stromatic masses of mycelium that form beneath the epidermis, frequently in the spaces beneath the stomata. The epidermis is ruptured as the conidiophores emerge. The bases of the tufts become more densely stromatic as they mature. (Fig. 6, A, B).

The obclavate or cylindrical conidia measure 18–60 × 5.4–10.8 μ and are 1- to 7-septate. The larger conidia are usually rather abruptly attenuated at the apex. In some instances the shorter 1- to 3-septate conidia are clavate rather than obclavate. The color is a pale brown and in some cases almost olivaceous. The conidia are produced singly. (Fig. 6, C, D).

Spermogonia develop during the fall.

DISCUSSION

It is clear from the literature review that 2 leaf spots may occur wherever peanuts are grown. That this fact has been generally recognized

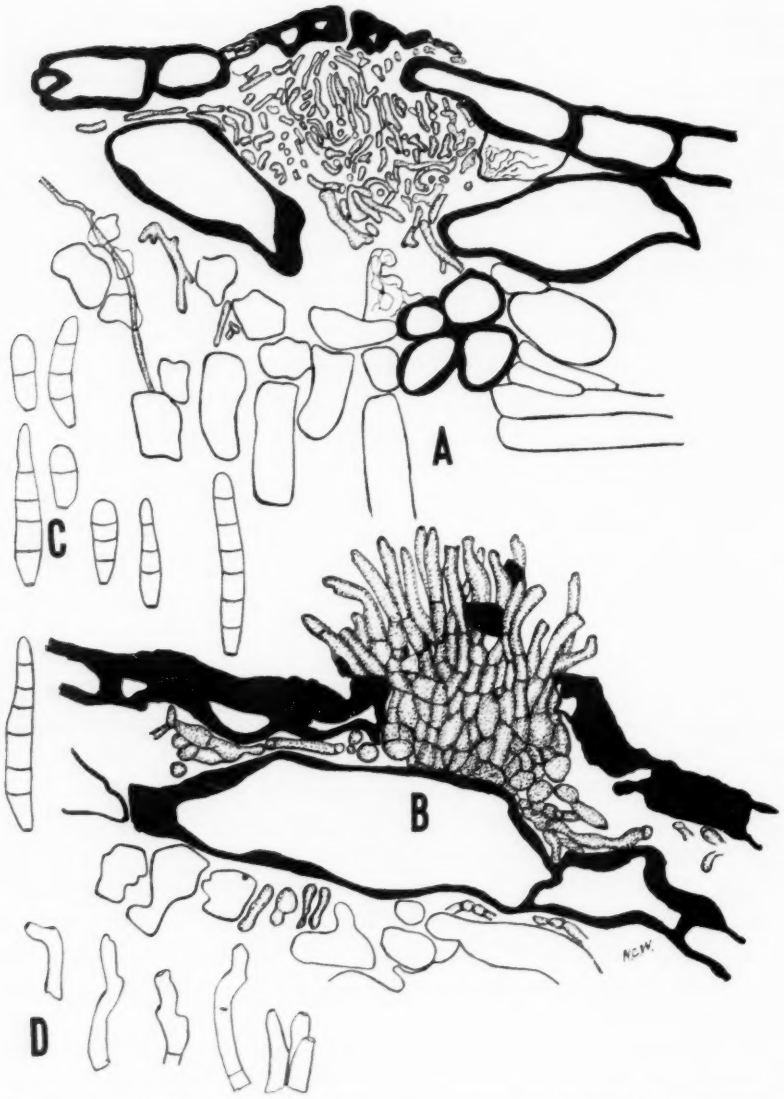

FIG. 6. *Cercospora personata.* ×540. A. Primordium of conidiophore tuft developing beneath a stomatal opening on the lower surface of a leaf. B. Mature conidiophore tuft showing the stromatic base. C. Conidia. D. Conidiophores.

without distinguishing the 2 has led to much confusion in the description and nomenclature of the causal fungi. Hori (13, p. 26) and Sawada (22) have described both leaf spots and their causal fungi and are the only writers who seem to have understood the confusion existing. Unfor-

tunately their descriptions are little known.[5] The purpose of this paper has been to direct the attention of pathologists and mycologists to the occurrence of 2 leaf spots on the peanut (*Arachis hypogaea* L.).

From the literature review and from observations at the Georgia Experiment Station it has been concluded tentatively that *Cercospora arachidicola* occurs rather consistently year after year, even when the weather is quite dry. *Cercospora personata* occurs at irregular intervals. In some seasons it is very severe, while in others few, if any, spots are to be found. In the 2 seasons in the last 5 years that *C. personata* has appeared at the Georgia Experiment Station it was undoubtedly the most destructive. More complete defoliation occurred in the years when *C. personata* was abundant. The conditions conducive to the development of *C. personata* are not well enough understood to attempt to explain why it appears only in certain years. Possibly wet seasons are more favorable to its development than the drier seasons.

SUMMARY

Two leaf spots have been found attacking the leaves of peanuts (*Arachis hypogaea*) in Georgia.

The nomenclature of the fungi attacking the leaves of peanuts is much confused.

A study of literature and a comparison of specimens from several collections have shown that there are often 2 leaf spots in nearly all peanut-growing areas, both sometimes appearing on the same leaf.

Cercospora personata (B. & C.) Ell. & Ev. has been generally accepted as the correct name of one of the fungi attacking the peanut. This fungus has abruptly obclavate or cylindrical conidia and the conidiophore tufts are concentrically arranged in the spots on the lower surface of the leaf.

Cercospora arachidicola Hori is probably the most common but is apparently the least destructive of the 2 fungi.

EXPERIMENT, GA.

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"BIG BUD," A VIRUS DISEASE OF THE TOMATO

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"Big bud" disease of the tomato was first described by Cobb (2) in New South Wales, under the name "rosette." The disease is now known by tomato growers in different localities in Australia under the names big bud, rosette, "blue top," or "bunchy top." For reasons given below, it has been considered advisable to adopt the name big bud as being the most generally satisfactory common name.

Since Cobb's first description of the disease, only short notes on it, with or without an illustration, have appeared in general articles on tomato diseases in Australian agricultural journals (3, 5, 9, 10, 12). In all the notes that have appeared to date, it is stated that the cause of the disease is not known, although in the New South Wales Farmer's Handbook (9) it is said that "the available evidence suggests that it is a disease of the virus type."

The experiments reported in the present paper confirm this suggestion and show that the disease can be transmitted by budding and grafting. No insect vectors have yet been discovered. The following account is presented mainly with the idea of calling attention to morphological and anatomical changes, which differ from those associated with any other virus disease.

NAME OF THE DISEASE

The name rosette, used by Cobb, has priority as far as date of publication is concerned, and it would have been retained if there had been no objections to it. These may be stated as follows:

(1) The name "tomato rosette" has since been used for other diseases. Selby (11) used the terms "potato rosette" and tomato rosette in the early part of the century for a disease that he attributed to *Rhizoctonia solani*, but his conclusions have not been generally accepted. Chupp (1) gives the name rosette for a tomato disease of unknown cause investigated by Orton and McKinney in Pennsylvania, but the report of this work is not available to us. From the description of symptoms given by Chupp, however, the disease is evidently different from the one dealt with in this paper.

(2) Although it is admitted that the final effect of the Australian disease is to give affected plants a dense, tufted habit, well described by the

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term rosette, this is not evident until a comparatively late stage. The most pronounced early symptom is an enlarged bladder-like calyx; and it seems advisable on general grounds to draw the attention of growers to early symptoms in preference to late. (Froggatt (5) even considered the early stage to be a separate disease, for which he proposed the name "Sheath-calyx.")

(3) It will become apparent from the description given below that a number of the symptoms are not paralleled in the rosette diseases of the virus type (peach rosette, wheat rosette, peanut or groundnut rosette) already described.

For these reasons the name big bud is proposed in place of rosette. It has the merit of being in fairly general use already, of drawing attention to the earliest symptom of the disease, and of avoiding confusion with the other tomato-rosette conditions. It also leaves the term rosette free in case one of these diseases is later found to be a definite entity requiring a name of this type. One disadvantage of the name, though not considered a serious one, is the fact that it has already been used for a disease of black currants in England caused by mites, which is often associated with the virus disease "reversion." There appears to be no connection between the big-bud conditions caused by the mites and the virus disease, however.

R. J. Noble and D. B. Adam, plant pathologists in New South Wales and Victoria, respectively, have been consulted, and also favor the adoption of the name big bud. The name bunchy top should be avoided and reserved for the disease described by McClean (8) from South Africa.

OCCURRENCE

As far as is known, tomato big bud does not occur in any other country than Australia, where it is found to some extent in all the States, but most frequently in New South Wales and Victoria. Usually only a very small percentage of plants are affected, and these occur scattered at random in the field. In South Australia, over a period of 10 years, no case has been seen where more than 5 per cent of plants have been affected, and the number is usually much smaller. In Victoria, Strickland (12) speaks of the results from manurial plots in 1930 being spoiled by the disease, which occurred to the extent of 10 per cent. In New South Wales, considerably higher proportions have been recorded at times. R. J. Noble, in a letter, speaks of cases where 50 to 100 per cent infection has been observed.

The prevalence of the disease varies from season to season, probably depending largely upon the abundance of some as yet undiscovered insect vector. It is predominantly a late-season disease, appearing in January or February, the midsummer and late summer months.

Plants affected with big bud may also become infected with spotted wilt or mosaic, and occasionally plants already infected with these diseases have been noticed to become affected with big bud.

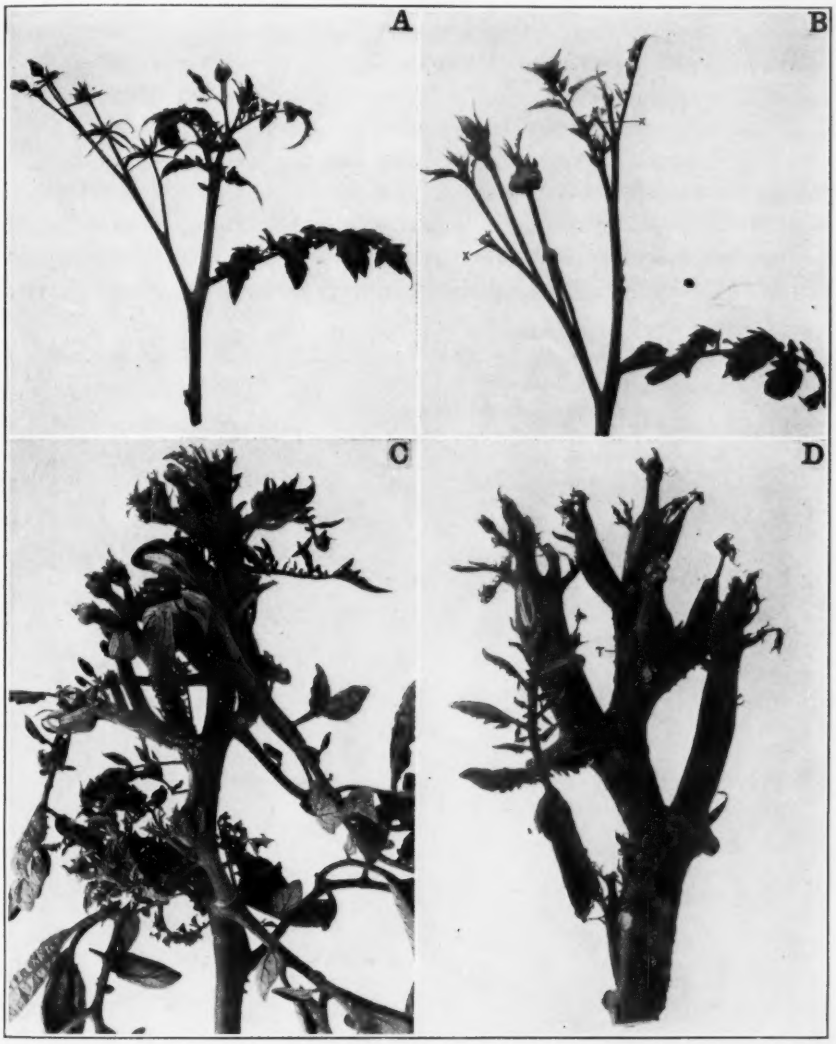

FIG. 1. A. Shoot of Dwarf Champion tomato with early stage of big bud. The flowers on the lowest truss were just opening when the virus invaded the top of the plants. B. Shoot of infected plant about 5 weeks after symptoms first visible. Note reduction of flower trusses to papillate branches at X. Both A and B from plants budded in glasshouse. C. Fairly late stage of big bud on a pruned Sensation plant from the field. D. Late stage on a pruned Marglobe plant from the field. Note that the branch T is morphologically a fruit truss.

SYMPTOMS

Plants affected with big-bud disease are characterized by some remarkable teratological growth changes, particularly in the flowering parts.

The Shoot. The first indication of infection appears at the tips of actively growing shoots. The youngest fruit truss, instead of becoming recurved as in normal plants, assumes an upright position (Fig. 1, A). The buds on the truss also point in a vertical direction, the calyx segments remain united almost to the tips, and the whole calyx enlarges to a form like a bladder with a toothed opening at the top. On pruned plants in the field the main shoot soon ceases growth, and the young leaves at the growing point fail to develop normally. A purple coloring due to the development of anthocyanin is often particularly marked along the veins of the bladdery calyces, on the under sides of the young leaves and on the youngest portion of the stem.

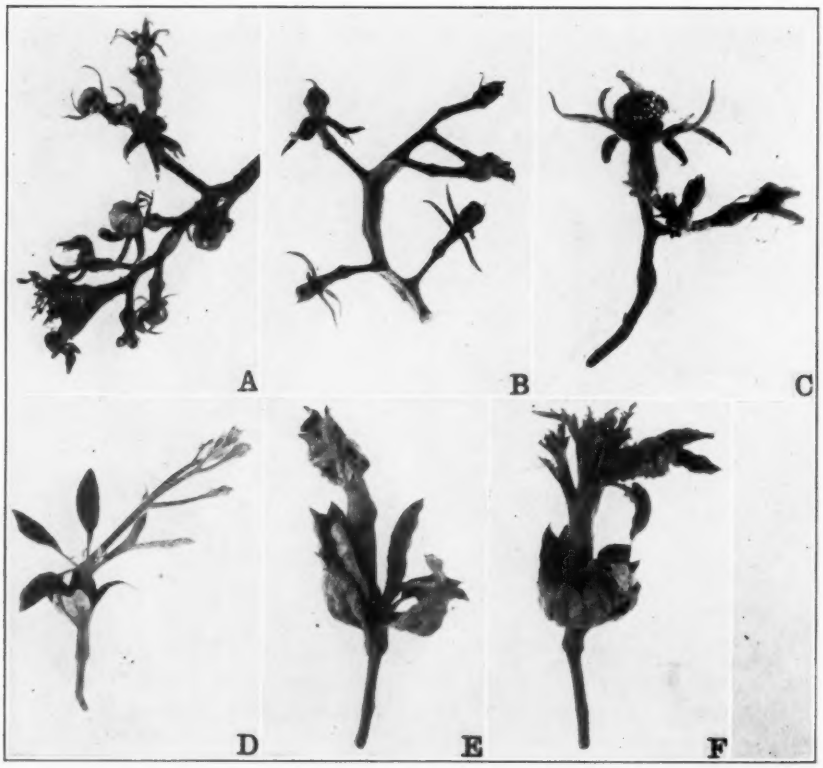

FIG. 2. A–C. Effect of the big-bud virus when it invades fruit trusses that have just set. Note arrest in growth of fruit, bursting of hardened pericarp exposing aborted ovules, thickening of pedicels commencing at abscisss layers, adventitious shoots on pedicels, and longitudinal splits exposing inner tissues. D–F. Different types of floral proliferation on tomato × currant-tomato crossbreds infected with big bud.

After a short time the axillary buds grow out, forming shoots affected in the same way as the main shoot. Simultaneously, there is a gradual thickening of the stems of the affected parts (Fig. 1, C) due to the formation of an abnormal tissue, described later, in close association with the internal phloem. In cases where the growth of the terminal buds completely ceases, the thickening of the stems may become very marked, and a diameter of 1½ to 2½ cm. be attained, even in what is morphologically a fruit truss. (Fig. 1, D.) Adventitious root initials break through the epidermis, and longitudinal splits may appear, laying bare the internal tissues in scars of characteristic form. (Fig. 1, D, and Fig. 2, B.)

On infected plants that are unpruned there appears to be more outlet for the increased growth activity in the development of numerous lateral shoots. The axillary buds of these lateral shoots grow out in turn, instead of remaining dormant, and the flowers proliferate as described below, so that the infected plant finally acquires a very tufted, rosette habit of growth. Partially pruned plants with a number of laterals already in this late stage are shown in figure 3, A and B.

In the greenhouse infected plants were observed to grow more rapidly than the uninfected at first, but after a few weeks they gradually ceased growth at the tips and acquired small purplish leaves. (Fig. 1, B.)

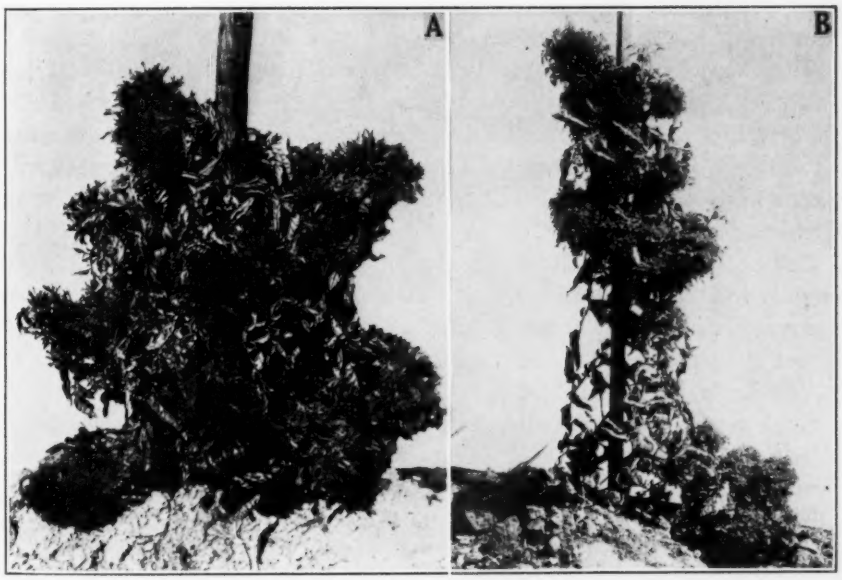

FIG. 3. Late stage of big bud on partially pruned field plants. A. At Bendigo, Victoria. B. On tomato × currant-tomato crossbred, Waite Institute, South Australia.

The Leaves. The first effect of the virus on young developing leaves may be to stimulate the formation of more small lateral leaflets from the main and secondary petioles. There may also be some upward rolling of the leaf margins. After some time the youngest leaves become yellowish green in color, particularly at the margins, the lateral leaflets are often much reduced (Fig. 1, B–D), and there is usually a purpling underneath due to the formation of anthocyanin in the cells of the lower epidermis. As the disease progresses, smaller and smaller leaves are formed, until in the dense rosettes that are finally produced the leaves may be only a centimeter or 2 long.

The Flower. Flowers that are fully formed at the time the virus invades the truss become erected from their recurved position but undergo little change beyond a slight virescence of the petals, appearing first along the midribs (Fig. 1, A). In younger flowers, just opening at this time, the corolla may become either stunted or enlarged and completely virescent. In both cases, there is a cessation of further development of anthers and ovary.

In buds at the end of the first infected truss and in all buds formed on subsequent trusses (Fig. 1, A) the sepals remain united and enlarge, so that the calyx swells up to the bladdery form previously described. Buds such as these never develop to normal flowers, and the central parts are either completely arrested in growth or proliferate in various ways, as follows.

There may be marked phyllody of the corolla, which frequently develops to a whorl of simple green petiolate leaves 1 to 3 cm. in length (Fig. 2, D–F). The insertion of this whorl of corolla leaves may remain close to the base of the calyx, the leaves scarcely or not projecting, or it may occasionally be carried up by an extension of the floral axis as much as 2 cm. above the calyx insertion (Fig. 2, D).

The anthers are usually inserted just above the foliaceous petals; they are pale in color and gradually die from the tip downwards without dehiscing. Owing to the thickness of the proliferated axis of the flower they are not syngenesious, as in normal flowers, but are inserted singly between the corolla leaves (Fig. 2, D). Occasionally single anthers are carried partially up the proliferated central axis.

The gynoecium is frequently carried up some 1 to 2 cm. on the proliferation of the central axis of the flower. In these cases, there is usually a distinct phyllody of the carpels, the individual leaves being either completely united to their apices or at least partially united by their margins and petioles (Fig. 2, E and F). In the former case, a transverse section of the gynoecium shows placental ridges bearing rudimentary ovules, in cavities with thin foliaceous outer walls. In the latter case, where there

is further development of the carpellary leaves, the extended axis of the flower gives rise to a system of short, thick-branched shoots some 1 to 2 cm. long (Fig. 2, F) with a characteristic dichotomous forking at the apices. Sometimes, however, the proliferating branches terminate in a second series of affected flower buds (Fig. 2, D). The proliferation of the central axis of the flower to a number of short-branched shoots may also occur without there being any sign of carpellary leaves developed.

The short shoots with dichotomous branching at the apices, which usually arise from the proliferated floral axis, bear minute colorless papillae at their tips (Fig. 4, A), with a few small leaf rudiments intermingled. They

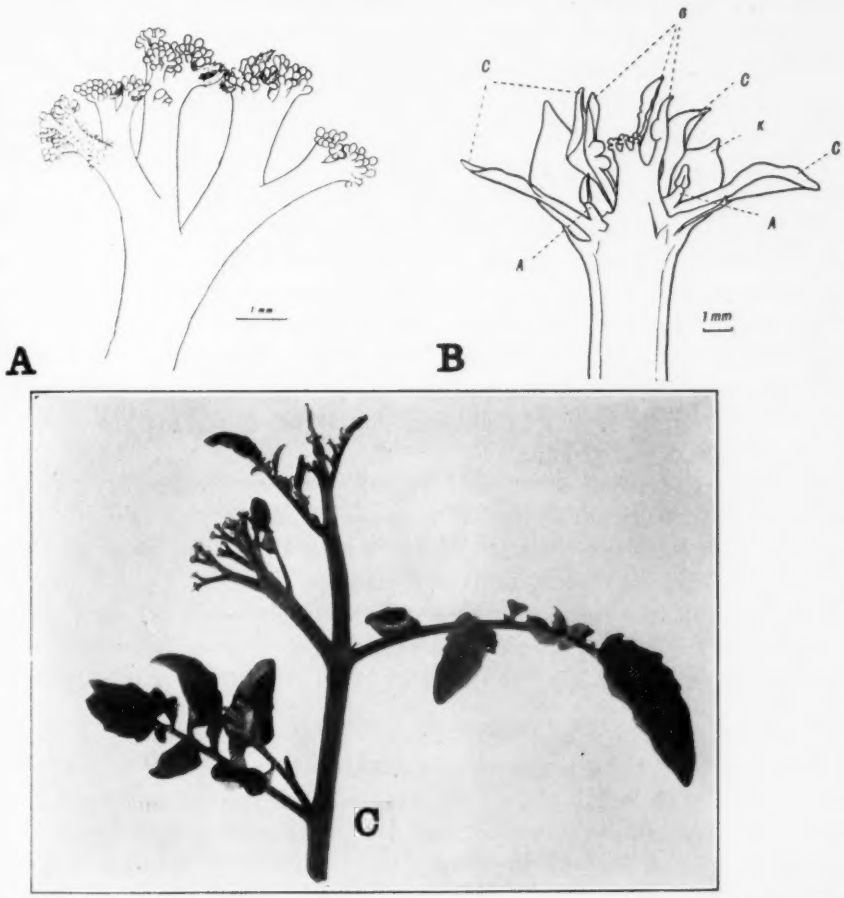

FIG. 4. A. One of the ultimate branches of a structure such as in C, showing papillae intermingled with leaf rudiments. B. Longitudinal section of a flower in which in addition to virescent petals and foliar carpels is a central structure with papillae. C. Reduction of flowering truss to a dichotomously branched structure with papillae at the tips, on a tomato × currant-tomato crossbred infected with big bud.

are usually strongly colored with anthocyanin. From occasional cases, such as that shown in Figure 4, B, where these papillae appear at the summit of a single short structure in the position of the gynoecium, the papillae appear to be homologous with ovules. There is no structural differentiation within them, however, and they later turn brown and die.

In later stages of the disease, the characteristic system of dichotomously branched shoots terminating in papillae frequently arises directly from the stem (Fig. 4, C) in such a position as to indicate that it represents a complete flower truss. In these cases it would appear as if the intermediate stage of flower formation had been suppressed and the final end of proliferation to this remarkably changed gynoecial structure attained directly.

The Fruit. Fruit already set when invaded by the virus is immediately arrested in growth (Fig. 2, A–C). In very young fruit there is sometimes a slow subsequent growth of the placental tissues that bursts the thin ovary walls and exposes the aborted ovules (Fig. 2, A). The latter may grow for a little while to greenish papillae (devoid of internal structure) projecting from the exposed placenta (Fig. 2, C). Meanwhile, the usual thickening of the stems characteristic of the disease appears on the younger parts of the truss, becoming most pronounced first at the abscise layer below each fruit (Fig. 2, A–C). Adventitious shoots may arise later from several places on the truss and grow and branch profusely in the usual way until they finally form a rosette of small leafy shoots. The adventitious shoots may arise not only from any part of the pedicels but from the aborted placental tissue, or even from between the arrested ovary and the calyx, in the position of the corolla.

Fruit that was well developed but still green at the time of infection becomes hard and tough and colors extremely slowly or not at all. In the younger fruit there may be a necrosis in the centers of the young embryos, which are embedded in a firm parenchymatous tissue with thick cellulose walls instead of in a tissue soon becoming gelatinous, as in normal fruit.

The Roots. No pathological changes have been observed in the roots of plants affected with big-bud disease.

ANATOMICAL CHANGES

The anatomical changes associated with the abnormal flower parts will not be dealt with in this paper. Here it is desired to call attention only to a very characteristic tissue that develops in association with the internal phloem in affected parts of the stem. In very much thickened stems, such as those in figure 1, C and D, this tissue is easily visible to the naked eye. When the stem is cut across it is seen as a band about 1 to 2 mm. across, internal to the wood, having a somewhat greenish, water-soaked appearance, owing to the close packing of the cells of which it is composed.

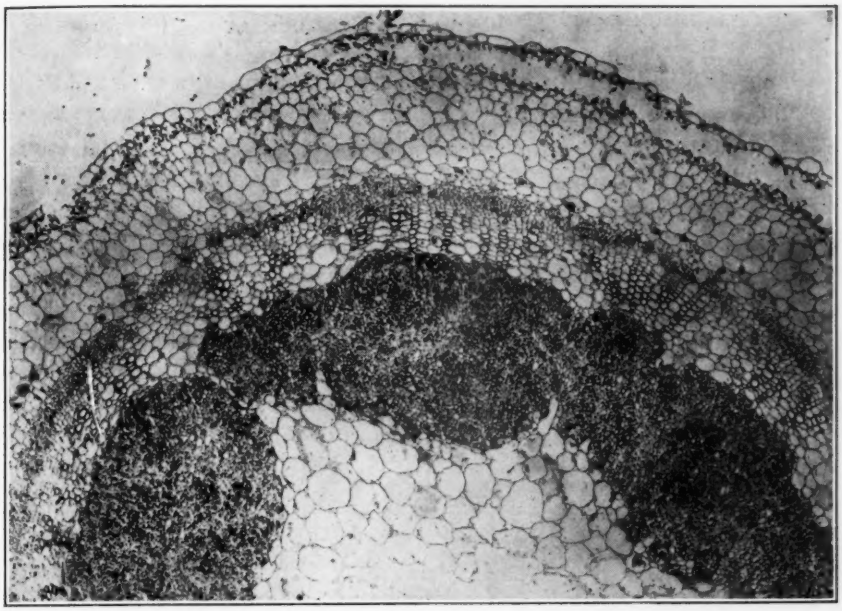

FIG. 5. Photomicrograph of a transverse section of a pedicel below an enlarged calyx. Note great development of adventitious tissue in place of internal phloem.

A transverse section from the pedicel below a bladder-shaped calyx is illustrated in figure 5. The great development of internal phloem tissue characteristic of the disease is immediately evident. The tissue is unlike normal phloem, however, in that the great bulk of it consists of small cells with prominent nuclei very similar to companion cells, that sieve tubes are rare, and that isolated tracheids are also found within it.

The tissue is found in all parts of the stem formed subsequent to infection by the virus. Arising first in scattered groups, resulting from the division of isolated pith cells, it soon increases and forms a continuous band. It is present also in considerable amount in the pedicels of affected fruit trusses and to a less extent in the petioles and midveins of the leaves. Wherever much of this tissue has been formed considerable amounts of starch are present in the pith.

No intracellular inclusions of the type described for certain mosaic diseases have been noted.

TRANSMISSION TESTS

Mechanical Transmission. No cases of successful mechanical inoculation of tomato big bud have yet been obtained. Freshly expressed juice from diseased field plants was used for inoculation to healthy tomato plants,

varying from seedlings to plants 15 in. or more in height. Inoculation was by rubbing the surfaces of a number of leaves, by needle scratching, or by injections of juice with a hypodermic syringe. The plants were kept from 5 to 9 weeks after inoculation, but all the experiments, which were done at various times, gave negative results. A number are summarized in table 1.

Budding and Grafting. Budding and grafting experiments, in which buds and scions from field plants diseased with big bud were used, have given successful transmission in every case in which the bud or scion remained alive. Transmission occurred even when the bud itself died and only a small pad of cortical tissue from the diseased plant lived and formed organic union. No transmission occurred in any case when the buds or scions died completely except in 3 cases in which grafted plants contracted the disease in spite of death of the scions. The scions on these grafted plants died very slowly, however, and it is possible that a small portion of

TABLE 1.—*Summary of transmission tests with big-bud disease of tomato*[a]

Date	Method of inoculation	Plants inoculated	Plants infected	Incubation period (days)	Notes
8.3.30	Rubbing + Needle Scratching	3 tomato 3 tobacco 3 *Nic. glutinosa*	0 0 0	....	Discarded after 38 days
13.3.30	Rubbing + Needle Scratching	5 tomato 5 tobacco 3 *Nic. glutinosa*	0 0 0	....	Discarded after 33 days " " 42 " " " " "
—.1.32	Rubbing	10 tomato	0	.....	Discarded after fruit set
17.3.32	Rubbing	24 tomato	0	.....	" " 65 days
17.3.32	Hypodermic	24 tomato	0	.....	" " " "
1.2.32	Grafting	10 field tomatoes	1	32	9 grafts failed
13.2.32	Budding	4 field tomatoes	4	31, 37, 37, 43	
18.2.32	Budding	16 field tomatoes	16	28–36	
8.3.32	Grafting	10 tomato	3	47–84	10 grafts but none took, 3 cases of transmission
8.3.32	Budding	15 tomato 4 tobacco 4 *Nic. glutinosa*	15 0 0	47–56	Repotted when pot-bound 6 other buds failed 2 other buds failed
17.3.32	Budding	6 tomato	6	56	Repotted when pot-bound

[a] All plants were kept in an insect-proof greenhouse except where it is definitely stated that field tomatoes were inoculated. Controls in the greenhouse and in the field, budded with healthy buds, remained healthy. There was no further natural spread of the disease in the surrounding field plants at the time the successful transmissions were obtained by budding and grafting.

tissue in a favorable position formed organic union during the first week or 2, but later died with the rest of the scion. The budding and grafting experiments are also summarized in table 1.

The incubation period varied from 28 to 56 days. The longer incubation periods were on older plants in the field or on plants that became rather potbound in the greenhouse. Many of the greenhouse tests were done on plants in 6-in. pots before it was realized that the incubation period was always long, and after some 3 to 4 weeks, when growth was obviously slowing up because of the small pots, the plants were transplanted to 8-in. pots and later developed the disease. It is considered that the average incubation period in plants kept growing under good conditions will be found to be somewhere near the minimum obtained in these experiments.

HOSTS OTHER THAN TOMATO

Plants of *Solanum nigrum* have been found near the tomato plots, almost certainly affected with the big-bud disease. Besides reduction in size of the leaves, and the presence of numerous axillary shoots, the flowering trusses were replaced by short branches with terminal papillae of the type described for the tomato disease. An adventitious internal phloem tissue similar to that found in tomato big bud was also present. In attempts to transmit the disease by budding to tomatoes, all the buds unfortunately died.

Four tobacco plants and 4 *Nicotiana glutinosa* plants were successfully budded with diseased tomato buds. No transmission to these plants took place, however, and flowering, which occurred some 14 to 15 weeks after budding, was quite normal in both species, the inserted diseased buds being still alive and a few of them having grown considerably.

DISCUSSION

The disease that presents the closest parallel to tomato big bud as regards effect on the host plant is undoubtedly cranberry false blossom, transmitted by the jassid *Euscelis striatulus* (4). A stimulation to the growth of axillary shoots, erection of the normally recurved pedicels, enlargement of the calyx lobes, virescence of the petals, abnormalities in stamens and ovaries, and frequent floral proliferation are all characters in common between the two diseases. Teratological changes proceed somewhat further in the tomato disease, however, in adhesion of the enlarged sepals, more marked changes in proliferation of the ovary, and particularly in the formation of the branches bearing papillae (Fig. 4).

Less marked floral changes, including virescence and proliferation, have been described for aster yellows and alloiophylly of anemones (6), but in these cases they are not invariably associated with the diseases. And, of

course, a stimulation to the growth of lateral shoots and a general upright habit of growth are characteristic of a number of virus diseases. One such disease, witches' broom of potatoes, has been transmitted to tomatoes (13). On the latter host, it has the following symptoms in common with tomato big bud: (1) the shooting of axillary buds, (2) marked dwarfing of the leaves at the growing points, (3) yellowing of the margins of the young leaves, and (4) purpling of the veins on the under sides of the leaves. That the diseases are distinct, however, is certain from the description of witches' broom on tomato given by Young and Morris (13). They state that their plants "bloomed profusely, and produced many small fruits; the buds and flowers developed normally." Their illustrations show flower trusses normally recurved, and there is a spindliness of infected stems rather than the thickening characteristic of tomato big bud.

The only parallel to the adventitious tissue that arises in association with the internal phloem in tomato big bud seems to be the phloem gall of Fiji disease of sugar-cane (7). This, however, is localized, so that numerous discrete galls are produced, whereas in tomato big bud the tissue soon forms a continuous thick band in all the new growth produced after infection, and the external phloem is not affected.

SUMMARY

Big-bud disease of tomato, originally named tomato rosette by Cobb, occurs in all Australian States, but most frequently in New South Wales and Victoria.

Abnormalities in the floral parts, involving reversal of the normal geotropic response, and various forms of over-growth, adhesion, virescence and proliferation are the most characteristic symptoms. The shooting of axillary buds and the formation of dwarfed leaves lead to a very tufted habit of growth in the later stages.

The stems of affected plants are somewhat thicker than those of normal plants, due to the formation of an abnormal tissue in association with the internal phloem.

The disease could not be transmitted by mechanical inoculation, but readily by budding and grafting, the shortest incubation period being 28 days.

The insect vectors of the disease have not yet been discovered.

Some field plants of *Solanum nigrum* showed symptoms similar to those of diseased tomatoes.

Buds from big-bud diseased tomatoes did not transmit the disease to tobacco or *Nicotiana glutinosa*.

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"SUSPECTED MOSAIC" OF THE STRAWBERRY[1]

E. F. GUBA

(Accepted for publication September 19, 1932)

In view of the interest being shown in this chlorotic leaf condition of the strawberry (Fig. 1), it may be timely to offer a brief account of the writer's observations in Massachusetts and of his efforts to determine the nature of this disease. Some of the information obtained confirms the results of other workers and is offered to support certain opinions as to the nature of the trouble.

This strawberry disease has already been studied by Berkeley (1, 2), Plakidas[2] (5), and Clark (3). Berkeley, who called it "suspected mosaic," was unable to produce the disease artificially. He found it to be seed-borne and systemic. According to him, it is confined to new seedling varieties, and does not spread in the field from one variety to another. The illustration accompanying Berkeley's article shows the chlorotic character of the disease and the pronounced dwarfing of a diseased plant. In Ontario, the symptoms appear with the unfolding of the leaves in the spring, disappear with the hot weather of early summer, and reappear again the following spring. These observations led Berkeley to entertain the feeling that the trouble was of genetic origin. Clark, who called the disease "non-infectious chlorosis," considered its connection with a virus of the mosaic type rather unlikely, because (a) the chlorotic leaves are not crinkled, (b) most, but not all, of the seedlings of chlorotic plants are chlorotic, and (c) all degrees of chlorosis prevail from apparently normal plants to those almost completely devoid of chlorophyll. The crinkling of the leaves, or a variation in the intensity of the symptoms, should not, however, argue more strongly for a genetic than a virus hypothesis. The crinkled condition of some of the foliage is quite characteristic of the disease (Fig. 1, A). According to Clark, it seemed probable that the condition was hereditary and was caused originally by a gene mutation, although further convincing evidence seemed desirable. Plakidas, like Berkeley, failed to produce the disease artificially with sap transfers, insects, or by reciprocally grafting diseased and healthy strawberry runners. He noted that a percentage of seed-propagated plants from chlorotic parents developed the disease the second year. Thus, very good published evidence makes doubtful the infectious nature of the trouble, but neither has the hypothesis of an hereditary condition been entirely convincing.

[1] Massachusetts Agricultural Experiment Station Contribution No. 152.

[2] Plakidas, A. G. Report on strawberry virus project. U. S. Dept. Agr., Bur. Plant Indust., Plant Dis. Rptr. **13**: 129–131. 1929.

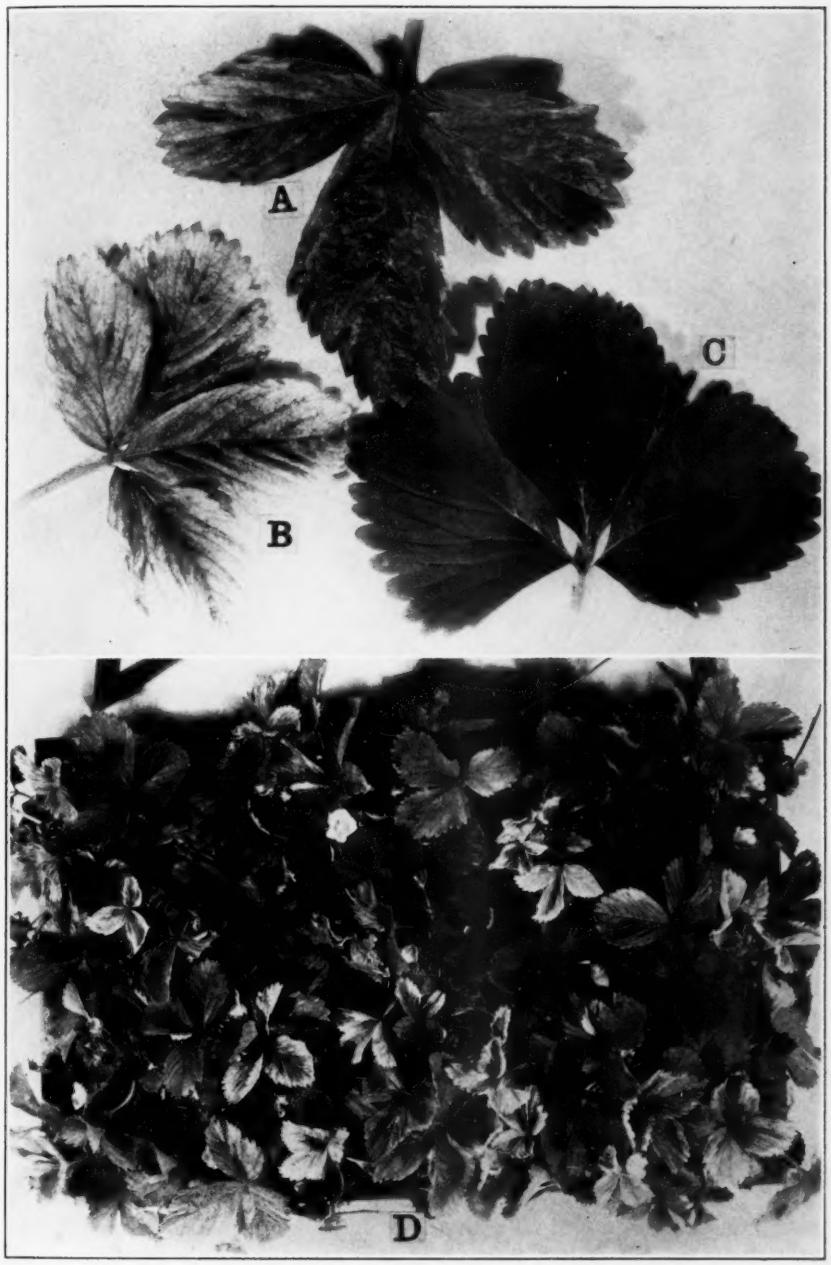

FIG. 1. Symptoms of strawberry yellows. A. Puckered and streaky-mottled leaf. B. Grayish white appearance around margin of leaf. C. Healthy leaf. D. Diseased plants of Howard 17 strawberry as they appear in the row in the field.

In Massachusetts this same disease referred to as "gold leaf" by Guba (4) is confined to the Howard 17 variety, expressing itself in plant degeneration and crop depreciation. In districts outside of New England it has been reported especially on Van Dyke, Eaton, some other new varieties, and many unnamed seedlings, none of which is grown in Massachusetts. Of particular interest is the fact that chlorosis is manifested in late April after the new beds are planted but disappears by the first week in July. This periodic appearance and disappearance of the disease is certainly not characteristic of viroses. The foliage, blossoms, and fruit show all stages of dwarfing, according to the intensity of the chlorotic condition, but no manifestation of so-called mosaic ever has been noticed in the fruit. Plants from a chlorotic source are much more difficult to establish in new plantings than normal plants, resulting in broken rows or skips (Fig. 2). There is also some mortality among chlorotic plants in the beds.

FIG. 2. Howard 17 strawberry bed in mid-June showing skips where severe chlorosis has resulted in plant mortality, and patches of chlorotic plants.

The chlorotic condition is quite general on the Howard 17 variety in eastern Massachusetts but its prevalence in strawberry beds is variable. In some beds it is general but in the majority of them its distribution is localized to one or more spots. Beds completely free of the trouble are uncommon.

In the writer's studies, seed from affected plants produced both chlorotic and heathy seedlings (Fig. 3), and the characteristic symptoms were manifested as soon as the seedlings developed their first true leaves. The tendency to dwarfing was very pronounced and ultimately the chlorotic seedlings died, their mortality being associated with almost complete absence of chlorophyll (Fig. 3). This inhibition in the development of chlorophyll is known to be characteristic of genetic chloroses in contrast to a degenera-

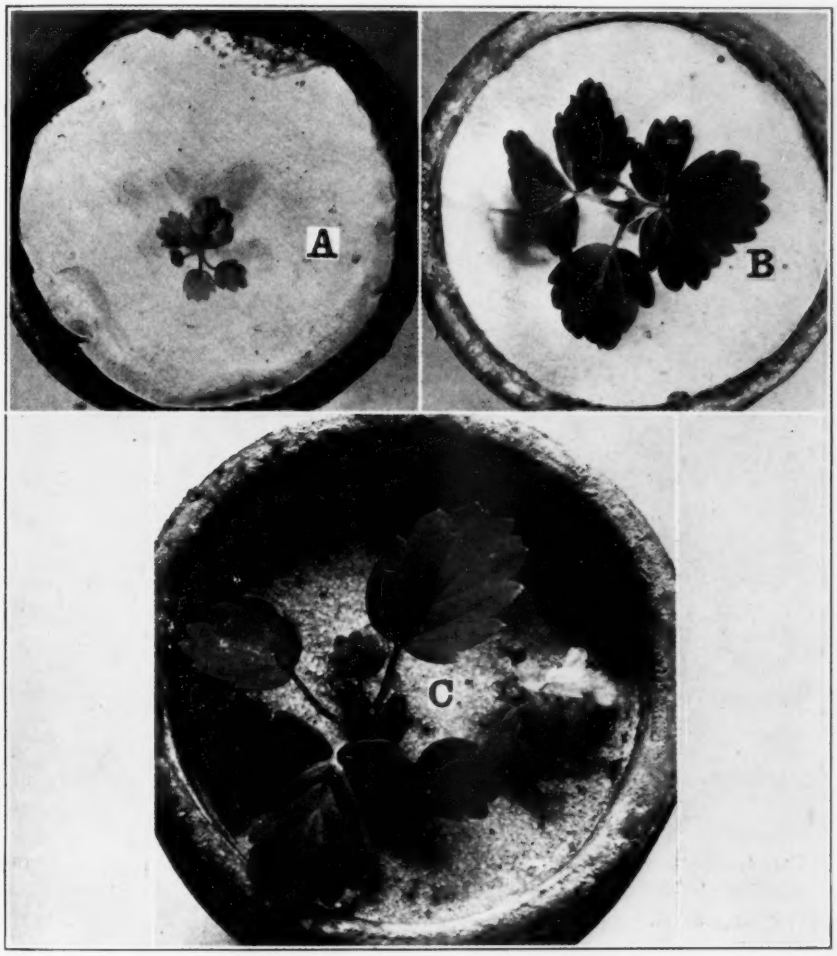

FIG. 3. Strawberry seedlings from a chlorotic parent. A. Chlorotic seedling. B. Normal seedling of same age as A. C. Normal and chlorotic seedlings of same age in same pot. The chlorotic seedling at the right is extremely dwarfed and almost entirely free of chlorophyll.

tion of chlorophyll as in viroses and is strong evidence in favor of a genetic hypothesis. Microscopic cross-sections of leaves from chlorotic and normal seedlings reveal a distinct contrast in the number of chloroplasts, (Fig. 4), and in the growth of the leaf tissues.

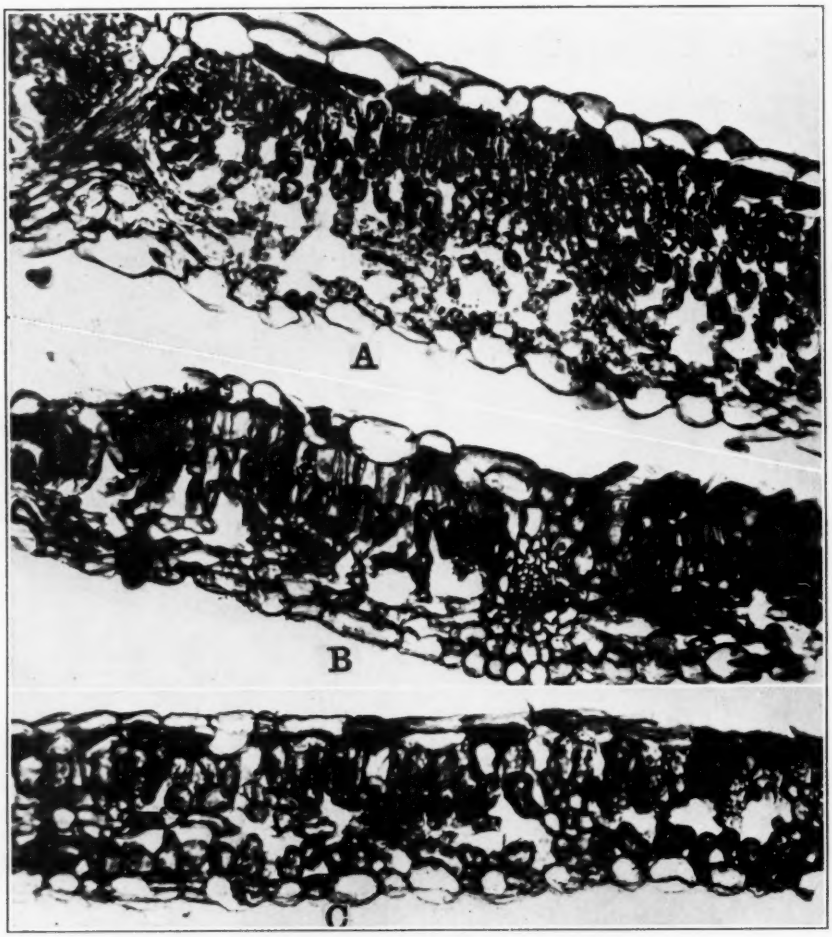

FIG. 4. Sections of normal and chlorotic leaves of strawberry seedlings. A. Normal leaf showing abundance of chloroplasts. B. Chlorotic leaf entirely free of chloroplasts. C. Chlorotic leaf showing a less complete inhibition in development of chloroplasts. × 150.

Efforts at artificial transmission of the trouble by possible insect vectors, notably aphids, white flies, tarnished plant bugs, leaf hoppers, and red spiders, and by inoculation with sap from diseased plants failed in all in-

stances.[3] Fertilizer dressings of iron sulfate, basic slag, dolomite lime, potassium permanganate, and manganese sulfate failed to effect any change in the expression of the trouble.

Certain observations in Massachusetts in the past few years would seem to throw further light upon the nature of the disease. A completely chlorotic bed was observed in the spring of 1932. This bed was planted in 1931 with seedlings that had come voluntarily from a normal green bed of Howard 17, which had been plowed under in 1930 because of rot in the berries. In previous years, when vegetative propagation was practiced, the grower had never observed the condition in his stock but when he resorted to sexual propagation it appeared suddenly. This observation is in agreement with the reports of Clark and Berkeley of instances of the sudden development of chlorosis among seedling varieties. The extent to which sexual propagation of strawberry plants is practiced is not known. Strawberry seedlings will grow where an old bed has been plowed under or where cull berries are dumped during the harvest season, and it is possible that some such plants have been used for propagating new beds. The practice of removing blossoms during the first year is by no means thorough enough to prevent the appearance of volunteer sexual plants in the rows. It is barely possible that some growers have used such plants in propagating from 2-year-old beds. If the hypothesis of a genetic condition is correct, sexual propagation would suggest an adequate explanation for the trouble.

The chlorotic condition is systemic. Where its prevalence is slight it does not become economically important unless a large number of daughter plants from chlorotic mother plants are used in the new planting. It is impossible for the grower to avoid these because the chlorotic condition does not appear until after the new beds are planted in the early spring. Further, in two efforts in 1930 and 1931 no success in the control of chlorosis attended selection and rogueing in the preparation of new beds when the vegetative stock used was originally from a moderately prevalent chlorotic source. In spite of these efforts, the disease was very prevalent in the spring of the bearing year.

In 1930 a bed of Howard 17 was set with daughter plants from a source showing a faint evidence of chlorosis. The chlorotic condition was not evident in the bed until the spring of 1931. In that season the chlorotic areas were definitely marked off. In 1932 spread appeared but it was too remote from the original chlorotic areas to have a systemic connection with plants showing chlorosis the previous season. Another bed was planted in 1931 with carefully selected chlorosis-free daughter plants from a bed in which the disease was present. During the spring of 1931 all plants suspected of

[3] Studies with insects were conducted under the direction of W. D. Whitcomb of the Department of Entomology.

showing chlorosis were removed. In the early spring of 1932 chlorotic daughter plants appeared to originate from normal green mother plants but, in the latter part of June, the chlorotic condition appeared generally in the rows.

These observations would suggest that these plants were potentially chlorotic but that the expression of the trouble was masked by the environment. It is difficult to believe that the trouble is due to an infectious virus in view of its behavior, of the negative evidence of transmission offered by Plakidas, Berkeley, and the writer, and of the fact that the condition has been developing among certain of the newer seedling varieties.

The manifestations of strawberry chlorosis are controlled or modified by the environment. Dry spring weather conditions seem to mask and modify the intensity of the trouble and to delay its appearance, while moist weather conditions favor its expression. Observations clearly indicate that potentially chlorotic plants do not always become chlorotic.

The disease is unquestionably of genetic origin and undoubtedly the result of a recessive genetic factor the expression of which is influenced by the environment. Where normal green plantings are being maintained the original heterozygous Howard 17 clon is undoubtedly being preserved by vegetative propagation exclusively. This clon should never be expected to show chlorosis so long as strictly vegetative propagation is practiced, although it would always bear the potential factor for chlorosis. Sexually propagated stock from this clon could show the trouble in varying amount, according to environmental conditions, and it is most likely that self fertilization rather than cross fertilization has contributed to the source of the trouble. According to this view, new varieties and seedlings genetically related to Howard 17 would always be liable to chlorotic outcropping.

If this hypothesis be correct, it is possible that some particular plant grower in eastern Massachusetts originally distributed sexually propagated plants of the original Howard 17 clon whence it has spread; and, since the disease is systemic, it has maintained and gradually increased itself vegetatively in these plantings. This hypothesis could account for the general prevalence of the disease in this section of Massachusetts in Howard strawberry beds. It would not be necessary to transfer chlorotic stock from locality to locality to spread the trouble, since, wherever the Howard variety is grown, some voluntary sexual reproduction with outcropping of chlorosis could occur. According to this hypothesis new varieties and seedlings containing the factor for chlorosis would always be liable to chlorotic outcropping and it would explain the distribution of the trouble among different varieties in the northeastern states and Canada, according to the survey report of Plakidas. Some particular instances of sexual propagation must

have occurred in eastern Massachusetts to account for the prevalence of the condition on the so-called Howard variety in this area.

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FURTHER STUDIES OF LIMA-BEAN SCAB

ANNA E. JENKINS[1]

(Accepted for publication August 30, 1932)

INTRODUCTION

New data on the geographic range and symptoms of the disease known as scab of Lima bean (*Phaseolus lunatus macrocarpus* Benth.) and on the life history of its pathogen tentatively identified as *Elsinoë canavaliae* Rac.[2,3] are here reported.

GEOGRAPHIC RANGE

The geographic range of the scab of Lima bean has been found to include Central America, the Dominican Republic, and possibly Jamaica, as well as the previously reported localities, namely, Cuba,[2] Puerto Rico,[2] and Sinaloa, Mexico.[4]

That the disease is more or less widely distributed in Central America and occurs there on the Lima bean growing both wild and cultivated was ascertained by a recent examination of dried specimens of *Phaseolus lunatus macrocarpus*[5] from El Salvador, Nicaragua, Costa Rica, and Guatemala, deposited in herbaria in the United States.

The specimen from Guatemala, 1890, now constitutes the earliest known record of the disease. Figure 1, A, shows an infected leaf of the Nicaraguan specimen, and figure 1, B, an infected pod of that from El Salvador.

[1] Acknowledgment is made to C. A. Weatherby, Curator of the Gray Herbarium, Harvard University, Cambridge, Mass., and W. R. Maxon, Curator of the U. S. National Herbarium, for fragments of specimens of *Phaseolus lunatus macrocarpus*, from Central America, showing lesions of Lima-bean scab. These have been deposited in the Mycological Collections of the Bureau of Plant Industry, U. S. Department of Agriculture.

[2] Jenkins, A. E. Lima-bean scab caused by Elsinoë. Jour. Agr. Res. 42: 13–23. 1931. (On page 20, paragraph 2, line 4, of this paper the word "here" should be changed to "where," and in line 7, "this structure was" be inserted after "structure").

[3] The Lima-bean scab organism is described as a distinct species in a related paper by S. C. Bruner and A. E. Jenkins to be published in the Journal of Agricultural Research, vol. 46.

[4] Jenkins, A. E. Additional data on the range and prevalence of Lima-bean scab. Phytopath. 21: 559. 1931.

[5] El Salvador, San Salvador (cultivated), 1925, Salvador Calderón 2250. In Gray Herbarium, Harvard University, Cambridge, Mass.

Nicaragua, Masaya, Jan. 27, 1903, C. F. Baker 117. Field notes: "A tall vine spreading thickly over low trees." In Gray Herbarium and in U. S. National Herbarium, Washington, D. C.

Costa Rica, San José, buissons au bord du rio Maria Aguilar, Nov. 27, 1894. A. Corduz 8964. In U. S. National Herbarium.

Guatemala, Guatemala, Feb., 1890. J. D. Smith 2297. In U. S. National Herbarium.

Information in regard to the occurrence of the disease in the Dominican Republic was secured through R. Ciferri. Sending representative specimens, he wrote[6] that as early as 1925 or 1926 he had observed this disease on leaves, stems, and green pods of the Lima bean but that on the basis of a superficial examination fungi were not apparent on the specimens collected and the disease was not diagnosed until the publication of the writer's[7] first report on it. Ciferri also stated that doubtless the disease is wide-spread in the Dominican Republic, that a large percentage of beans offered for sale in the interior markets are affected, and, further, that the disease is particularly common in the wet districts of the northern and southern parts of the Republic and rare in semixerophytic zones, as at Santo Domingo.

The record of the possible occurrence of the disease in Jamaica is based on the fact that scabbed Lima beans, said to have come from that country, were recently intercepted in a passenger's baggage by a port inspector of the United States Plant Quarantine and Control Administration.

Inquiries have been made in regard to the occurrence of the disease in South America and in West Indian Islands other than those already mentioned, but thus far no record of any Elsinoë disease on Lima bean in these regions has been obtained. As yet no record of the occurrence of Lima-bean scab in the Orient is available.

The continued prevalence of the disease in Cuba is indicated by the fact that typically spotted pods have been frequently intercepted during the shipping season, November 1, 1931, to March 31, 1932, by inspectors of the Plant Quarantine and Control Administration of the United States Department of Agriculture, at the Port of New York. The disease has also been common on Puerto Rican Lima beans received at the same port during the period named.

In the summer of 1931 intensive search for scab was made in commercial Lima-bean fields in Georgia, South Carolina, North Carolina, Virginia, New Jersey, and New York, by J. H. Miller and B. S. Crandall, both at that time of the Plant Disease Survey, and by W. D. Moore, of the Division of Horticultural Crops and Diseases, Bureau of Plant Industry. The writer examined specimens of leaves, stems, and fruits received from these investigators and other American-grown Lima beans secured in markets at Washington, D. C., and elsewhere in the East. No trace of the disease was found on these American-grown beans. W. A. McCubbin, of the Plant Quarantine and Control Administration, surveyed Lima-bean-growing regions in California in 1931 but did not find the disease there.

[6] Letter dated Nov. 21, 1931.

[7] Jenkins, A. E. *Loc. cit.* (Footnote 2).

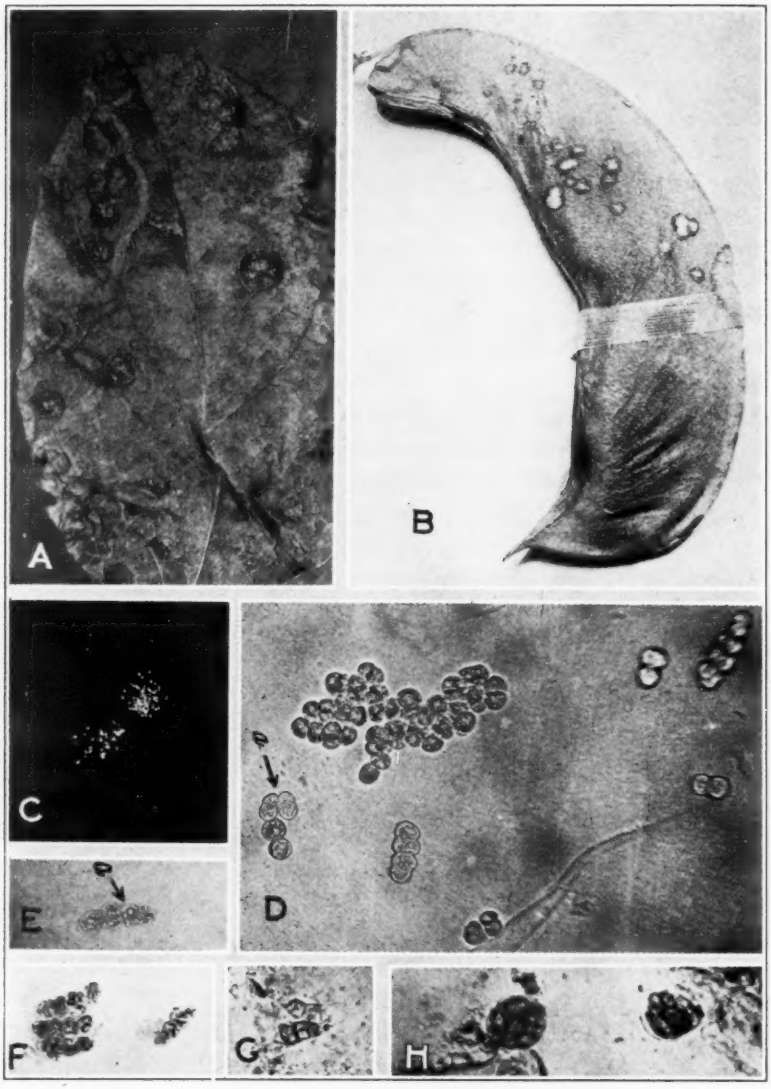

FIG. 1. A and B. Scab lesions on dried specimens of Lima bean (A) from Nicaragua, 1903 (×about 2) and (B), El Salvador, 1925 (×about 1). C–F. Cultures of the Elsinoë causing the disease from ascospores discharged from pod lesions to corn-meal-agar surface. ×380. D, *a*. Swollen sprout conidia. E, *a*. Refringent granules. G and H. Reddish-brown ascospores from pod lesions, those in H within ascus. ×380. For further explanation, see text.

SYMPTOMS

During the shipping season of November 1, 1930, to March 31, 1931, further studies showed that, while scab lesions may be noticeably swollen

or thickened, they are not the cause of the projections on pod margins, although they may sometimes be found on these regions as well as on other parts of the pod. The earlier material had seemed to indicate that such development resulted from attack by the fungus, although, even then, jaggedness of the pod margins was found independent of the lesions. The same phenomenon also has been noticed by S. C. Bruner in connection with his study of the disease.

LIFE HISTORY

In order to obtain some data on the longevity of the Elsinoë pathogen of Lima-bean scab, tissue cultures were made early in April, 1930, from fresh, diseased Lima beans from Cuba, received through the Plant Quarantine and Control Administration. Late in August, 1930, additional tissue cultures were made from the dry pods, some of which had been kept at room temperature and others at 17° C. The fungus was isolated only from the fresh pods. This result suggests that the pathogen was not viable on the dry pods, which were nearly 5 months old.

While the pods just referred to were fresh, additional cultures were made from them, mainly to determine whether ascospores would become distinctly colored with age. Ascospores for observation were obtained by placing pod lesions so that the ascospores from them could be discharged upward (less than half an inch) to water on a glass cover slip and to corn-meal-agar medium on glass slides.

After the spores had been discharged on these media, the cultures were placed in Petri dishes lined with filter paper and were held at a temperature of about 8° C. When placed at this temperature the ascospores, then not noticeably swollen, had in some cases begun to sprout conidia from their surfaces. Twenty-four hours later in the water culture and 5 days later in the agar culture both ascospores and conidia had become considerably swollen. In the agar culture, conidia were sometimes seen lying in a row at the side of an ascospore, evidently in the position in which they were produced. Figure 1, D, shows the appearance of ascospores in the agar culture when 8 days old, and figure 1, E and F, their appearance when 30 days old. In the older cultures the ascospore contents were glistening and coarser than in the younger cultures, and in some cases they appeared greenish. (Fig. 1, F). In no instance, however, did they show any deeper color than pale yellow, in either the water or the agar cultures. Petri-dish cultures, corresponding to those just described but grown at room temperature, served only to show that the inoculum was purely of the Elsinoë, for growth took place too rapidly for continued observation of the ascospores. The colonies when 7 days old were readily visible (Fig. 1, C) and were covered with conidia.

In another experiment fresh pod lesions were held for a few days in a moist chamber, and ascospores from them were then examined for coloration. These ascospores were reddish brown (Fig. 1, G and H), the coloration possibly having been derived from the pigment in the lesions. Similar observations have been reported for Myriangium[8] and Uleomyces.[9] Further study of the Alternaria commonly associated with the Elsinoë on its natural substrate shows that its conidia are sometimes easily confused with the colored muriform ascospores of the Elsinoë. A colored muriform spore, once thought[10] to be an ascospore of the Elsinoë, is now believed to be an Alternaria conidium and not one of the colored muriform Elsinoë ascospores of about the same size present on the same slide.

In order to obtain some data on the temperature relations of the Elsinoë, test-tube slant cultures of the fungus on potato-dextrose-agar medium were grown at constant temperatures of 0°, 5°, 10°, 15°, 20°, and 25° C. The inoculum for each culture consisted of a small amount of cultural growth about 0.5 mm. in diameter. After 3 weeks, the 2 sets of cultures, each made from a different isolation, showed no growth at 0°, slight growth at 5°, and increasing amounts of growth up to 25° C.

SUMMARY

Costa Rica, Nicaragua, Guatemala, and El Salvador, in Central America, the Dominican Republic, and possibly Jamaica are added to the known range of Lima-bean scab, previously reported from Cuba, Puerto Rico, and Sinaloa, Mexico.

Although lesions on pods may be noticeably swollen or thickened, they are not the cause of projections on pod margins, as previously reported.

Failure to isolate the pathogen of the disease from diseased dry pods held for nearly 5 months, some at room temperature and others at 17° C., suggests that the fungus was no longer viable on this material. Ascospores removed from pod lesions to culture media (water and corn-meal agar) and held at about 8° C. to check growth remained hyaline or pale even when a month old, whereas ascospores in fresh pod lesions kept moist for a few days were reddish brown, possibly deriving their color from pigment in the lesions. Cultures grown for 3 weeks at the constant temperatures of 0°, 5°, 10°, 15°, 20°, and 25° produced no growth at 0°, slight growth at 5°, and increasing amounts of growth up to 25° C.

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[10] Jenkins, A. E. *Loc. cit.*, pl. 5, C. (Footnote 2).

NEW LOCATIONS FOR PHYTOPHTHORA CITROPHTHORA AND P. HIBERNALIS ON CITRUS[1]

HOWARD S. FAWCETT[2]

(Accepted for publication August 31, 1932)

Phytophthora citrophthora (Sm. and Sm.) Leonian has been found recently in Florida and Louisiana, and *P. hibernalis* Carne, in California.

During a trip to Florida in January, 1932, occasional examples of a decay of oranges and grapefruit resembling citrus brown rot were noticed, but only on fruit that was lying on wet soil under trees at the edge of swamps or drainage ditches. Isolation culture tests gave pure cultures of *Phytophthora citrophthora* in three widely separated districts in Florida and cultures of *P. parasitica* Dastur in one other district.

Phytophthora citrophthora was obtained at Wildwood, Vero Beach, and Palmetto, Florida, and at Buras, Louisiana. Near Wildwood the oranges from which the fungus was obtained had been lying on moist soil in a large hammock grove where the orange trees were growing among the native oaks and palmettos and on the edge of swampy land. Near Vero Beach and Palmetto, grapefruits from which the fungus was isolated had been lying on very moist soil at the margins of drainage ditches.

In Louisiana, where heavy rains had been recent, the same fungus was isolated many times from oranges showing brown rot. Some of the fruits were obtained from the orchard and others from the packing houses in the vicinity of Buras in the delta region below New Orleans.

The cultures of *Phytophthora parasitica* were obtained from orange fruits with a decay resembling brown rot, on wet soil near Courtney, Merritt's Island, Florida.

The best method of obtaining a pure culture from these fruits was to take out a seed aseptically by means of sterilized scalpels, tweezers, etc., and to drop this seed on a slant tube of glucose-potato or corn-meal agar. If the fruit was seedless a piece of the albedo, core, or inner membrane was used.

These isolations from Florida and Louisiana were compared by growing them in parallel cultures with known strains of *Phytophthora citrophthora* and *P. parasitica* on corn-meal and glucose-potato agar. They were then grown in dilute prune extract, a bit of the mycelium transferred to tap water in shallow dishes and in from 24 to 48 hours (at 18° to 20° C.) all of them formed abundant sporangia. All of them appeared to be typical *P.*

[1] Paper No. 273, University of California Graduate School of Tropical Agriculture and Citrus Experiment Station, Riverside, California. Published with the approval of the Director of the Station.

[2] Professor of Plant Pathology.

citrophthora except the isolation from Courtney, Merritt's Island, Florida. Lemon and orange fruits were then placed in the water of the shallow dishes containing these sporangia and typical brown rot was produced in from 3 to 6 days from all isolations. The brown rot produced by *P. parasitica* usually tended to be a little lighter in color than that due to *P. citrophthora* but was very similar otherwise. These isolations were sent to Dr. S. F. Ashby of the Imperial Mycological Institute, who confirmed the author's identification of these strains. This appears to be the first authentic record of *P. citrophthora* in Florida.

Orange fruits having a decay resembling brown rot had been noted in Florida before, once by H. J. Ramsey and once by H. R. Fulton. The latter isolated a Phytophthora from two grapefruits collected at Orlando, Florida, and concluded that it was *P. parasitica*.[3]

The occurrence of *Phytophthora citrophthora* has now been reported in many citrus-growing countries of the world. The fungus was first described and reported by Smith and Smith[4] in California in 1906. It has since been reported in Arizona by Fawcett, Brazil by Averna-Sacca, Chile by Opazo, Union of South Africa by Doidge, Australia by Carne and others, Spain by Fawcett, Italy by Petri and by Fawcett, Egypt by Sirag-el-Din, Palestine by Reichert, Hawaii by Pope, Japan by Sawada, and the present report adds Florida and Louisiana.

In February, 1932, following the snows and rains of January, attention was called to the prevalence of brown rot on orange trees in the Cucamonga district of California. Not only were the fruit and leaves attacked in the usual manner, *i.e.*, up to a height of 3 feet from the ground, but frequently places were noted where the attack was much higher, occasionally nearly to the top of the trees. It was suspected that some other Phytophthora was present, and not the usual brown rot fungus. Although the appearance of the decay resembled that produced by *P. citrophthora* and *P. parasitica* (the organisms most commonly associated with citrus brown rot in California), isolation culture tests showed that another Phytophthora was present. This was sent to S. F. Ashby, who identified it as *P. hibernalis* Carne.[5]

This appears to be the first definite record of this fungus on citrus in America. This species was first described by Carne in western Australia. It has been found also in other parts of Australia. It was determined by

[3] Fawcett, H. S., and H. Atherton Lee. Citrus diseases and their control. P. 373. McGraw Hill, New York, 1926.

[4] Smith, R. E. and E. H. A new fungus of economic importance. Bot. Gaz. 42: 215–221. 1906.

[5] The author is indebted to Dr. S. F. Ashby of the Imperial Institute of Mycology, Kew, Surrey, England, for identification of this fungus and for confirmation of the identity of the isolations of *Phytophthora citrophthora* from Florida and Louisiana.

Carne to be the same as that reported as *Phytophthora* sp. by Monz da Maia in Portugal. Later, Bensaude reported the same fungus on both leaves and fruit in Portugal. This species is remarkable in having a very low optimum temperature for growth of about 12° C. as recorded by Carne. It is significant in this connection that the occurrence in California followed closely a period of cool weather with an unusual amount of snow and with frequent rains. Cultures of this fungus have been found to die in a short period at a temperature of 25° C. This heavy attack of the leaves near the ground on orange trees has been noted by the author in previous years and he believes now that *P. hibernalis* may have been in California for a number of years without having been recognized. It is also remembered that a similar fungus was isolated at Oroville, California, in a very cold spring, about 1922, and that the fungus soon died at room temperature of summer (about 25° C. at Riverside) before it could be studied. It is now believed that this isolation was *P. hibernalis*. This fungus is so sensitive to the ordinary temperatures of late spring and summer in California that it could presumably not exist except at some depth in the soil or by means of the resistant oospores during those seasons of the year.

Adding the above information to that previously reported, there are now known to be on citrus fruits in California at least three species of Phytophthora, all capable of producing citrus brown rot. These are *Phytophthora citrophthora*, *P. parasitica* (*P. terrestris*), and *P. hibernalis*.[6] These three have also been reported in the Mediterranean region. Two of these, *P. citrophthora* and *P. parasitica*, appear to have a wide distribution, having been reported already in most of the other citrus-growing districts of the world, including Florida. Other species of Phytophthora that have been reported so far on citrus are *P. citricola* in Japan, *P. palmivora* (*P. faberi*) in the Philippines and West Indies, and *P. arecae* in India.

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[6] A Phytophthora isolated from roots of citrus in Tulare County, Calif., has been identified also by Dr. Ashby as probably *P. megasperma* Drechsler.

PHYTOPATHOLOGICAL NOTES

An Apparent Case of Transmission of Cranberry False Blossom through a Natural Graft.—All attempts to transmit false blossom of the cultivated cranberry, *Vaccinium macrocarpon* Ait., by methods other than insect transmission with the leaf hopper, *Ophiola striatula* (Sleesman) Fall., as the vector have failed. The usual methods for artificially transmitting virus diseases, such as pricking or scratching with a needle, rubbing or other method of leaf mutilation, and even injecting virus by means of a hypodermic needle have produced no results. Some other virus diseases are similar to false blossom of the cranberry in this respect. Thus, Kunkel[1] mentions various methods used to transmit aster yellows artificially and says: "All attempts to transmit aster yellows mechanically, except by budding, have failed." Peach yellows also cannot be transmitted artificially by any method of rubbing, puncturing, or injection, although it is readily transmitted by budding or grafting. The only known attempt to transmit false blossom by grafting is that reported by Dobroscky,[2] who states that: "Grafting experiments were attempted several times. Cranberry stems are very woody, wiry and thin. Inarching grafts were made but the plants did not grow together. No infection resulted even though the stems were in external contact for long periods of time. If the grafts had been successful infection might have resulted."

The discovery of a natural graft involving apparent transmission of false blossom is, therefore, of considerable interest. The natural graft was found among some young vines of the Wales Henry variety that had been grown on the State bog at East Wareham, Massachusetts, from stem cuttings planted in May, 1931. In preparation for planting, the old vines were removed from several square yards of the bog and a layer of fresh sand, to a depth of about 2 in., was placed over the peat substratum. The cuttings were made from selected apparently healthy vines, although it was found later in the summer that a number of the cuttings had been made from infected vines. The cuttings, 6 to 8 in. long, were inserted singly in holes to a depth of about 2 in. into the peat below the sand covering. In plantings as ordinarily made 2 or more cuttings are inserted in a hole. In one instance, here described, 2 cuttings happened to be inserted together so that the stems were crossed, forming an X. One of the cuttings (Fig. 1, A) was apparently from a vine infected with false blossom, as the plant from

[1] Kunkel, L. O. Studies on aster yellows. Amer. Jour. Bot. 13: 646–705. 1926. See pp. 679, 681.

[2] Dobroscky, Irene D. Studies on cranberry false blossom disease and its insect vector. Contr. Boyce Thompson Inst. Plant Res. 3: 59–83. 1931. See p. 64.

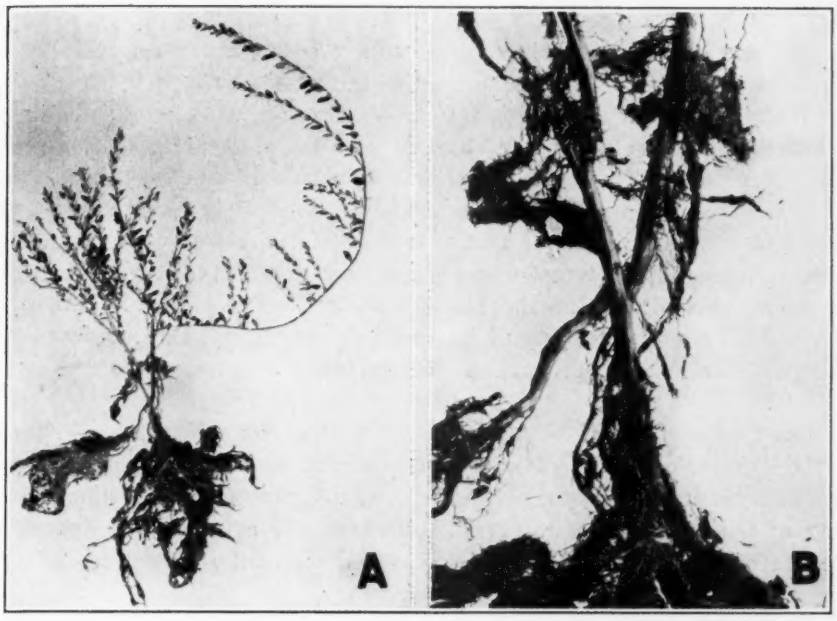

FIG. 1. A. Two young cranberry plants united by a natural graft. False blossom has apparently been transmitted to the plant at the right from the originally diseased plant at the left through the graft. About $\times \frac{1}{4}$. B. Closer view of the naturally grafted stems. $\times 2$.

this cutting showed throughout marked symptoms of the disease and produced 1 flower that was very characteristic of the disease. The other cutting (Fig. 1, B) seems to have been healthy when it was set out. This plant showed very definite infection on several uprights from a runner near the base of the plant. Branches above this runner, the distal portion of the runner itself, and 1 upright from it, most remote from the point of origin of the runner, showed no evidence of infection. From this it seems probable that the second plant was infected sometime during the season of 1931, as in false blossom of the cranberry evidence of infection generally does not appear until the year following that in which infection occurs. The relative position of diseased and healthy portions of the second plant also indicates that the infection came from below, *i.e.*, from the direction of the line of union in the graft. Healthy and diseased cuttings in the same hill have been noticed at various times by different observers[3] and often grow for long periods of time without the disease spreading from one to the other. The occurrence of a natural graft in any such case has, heretofore, been unknown.

[3] Personal communications from Neil E. Stevens and from H. F. Bain.

There is, of course, a possibility that the cutting that developed symptoms of false blossom in 1932 may have been made from an infected vine, as was true of some other cuttings in the small area of new planting of the Wales Henry variety. This variety is very susceptible to false blossom, and the vines from which the stems were selected for propagation were badly infected with the disease. For this reason it was very difficult to select vines for propagation that could be known certainly to be free from false blossom. However, the fact that none of the vines grown from cuttings developed symptoms of false blossom, except those in which the disease appeared in 1931, makes this possibility negligible.—H. F. BERGMAN and W. E. TRURAN, Cranberry Disease Field Laboratory, U. S. Department of Agriculture, East Wareham, Massachusetts.

Two Hosts of the Pokkah-bong Disease other than Sugarcane.—Early in the summer of 1929 a disease, apparently very similar to pokkah bong, was observed on several young corn plants. A microscopic examination of some of the affected tissues revealed the presence of numerous Fusarium spores that were very similar in size and shape to the spores of the Fusarium causing pokkah bong on sugarcane.

A short time after the affected corn plants were observed a similar affection was noted on young sorghum plants growing close by, and these also, when examined under the microscope, showed the presence of Fusarium spores. The symptoms on the corn and sorghum plants were very similar to those on sugarcane caused by pokkah bong and strongly suggested that these plants might be additional hosts of the pokkah-bong fungus.

On neither the corn nor the sorghum were the chlorotic spots quite so pronounced as on sugarcane. The leaf bases on the corn were more of a yellow chlorosis, but the red and black markings were almost identical with those observed on cane. In some cases a top rot had developed, which had the same obnoxious odor as the top rot on sugarcane. Many cases were observed where the corn tops were distorted and curved and nearly always, on these plants, the lesions or cavities could be found on the surface of the stalks. (Fig. 1, A.) No infection has yet been found inside the corn stalks.

Infection on the sorghum plants, while usually not so severe, is very similar to that observed on sugarcane. The leaf bases show the chlorotic spots and red markings very plainly, and, where the red markings dry and fall out, the leaves have the same ragged appearance as the similarly affected sugarcane leaves. (Fig. 1, B.) The red markings on the sorghum leaves are much more pronounced than on sugarcane, but this is to be expected, as most all sorghum leaf diseases are accompanied by a distinct reddening of the immediate or surrounding tissues. No infection has been observed inside the sorghum stalks, but, on the more susceptible varieties

FIG. 1. Pokkah bong. A. Produced by inoculation of young corn plants. Note distortion and rot on some plants. B. On sorghum. The ragged appearance is caused by the affected tissues drying and falling out.

and under favorable conditions, the fungus might penetrate to the inner tissues of the stalks.

Numerous isolations have been made from infected plants of both the corn and sorghum, and in nearly every case a Fusarium fungus was obtained. This was isolated in pure culture and inoculated back onto several corn, sorghum, and sugarcane plants. The symptoms produced on the corn and sorghum from these inoculations were identical with those from natural infection, while those on the sugarcane were typical of pokkah-bong infection. Pure cultures were then obtained from sugarcane plants affected with pokkah bong and inoculated onto several corn, sorghum, and sugarcane plants. These inoculations produced almost exactly the same symptoms as were obtained from the inoculations with the Fusarium spores isolated from the corn and sorghum.

These experiments were repeated several times with similar results in each case. The fungus isolated from sugarcane plants affected with pokkah bong produced typical pokkah-bong symptoms when inoculated onto corn and sorghum, while the fungus isolated from the affected corn and sorghum plants produced similar symptoms when inoculated onto sugarcane. It is very evident from the results of these experiments that the pokkah-bong disease of sugarcane is capable of attacking both corn and sorghum and that these plants are additional hosts of the pokkah-bong organism.—C. N. PRIODE, Cuba Sugar Club Experimental Station, Central Baraguá, Cuba.

Cuban Streak.—In the fall of 1931 a peculiar type of striping was observed on the leaves of a single stalk in a stool of POJ 2725 sugarcane growing in the experimental plots of the Tropical Plant Research Foundation at Central Jatibonico. The stripes were unlike those manifested by any other cane disease I have had under observation in Cuba and appear to be those of a disease new to the island. For convenience in describing, it has been named "Cuban streak." At the time the disease was first observed a careful inspection was made of all the surrounding plants in the field, but no other cases of infection were found. A few weeks later a similar striping was observed on a few plants of the CAC 323 variety growing close by. The field has been inspected several times since, but no new infections have been discovered.

The stripe-affected plants grew in a field that was surrounded by cane showing a high percentage of mosaic infection. The symptoms of Cuban streak, however, were very different from those of the mosaic, in that they occurred as distinct, chlorotic stripes with no indication of mottling, such as is characteristic of sugar-cane mosaic. It was first thought that the malady might be the same as the corn stripe disease, but a comparison of diseased leaves from corn and cane plants showed the symptoms to be very dissimilar.

The dissimilarity of symptoms, however, might be due to a difference in the host manifestation when the disease passes from corn to cane, and later experiments may prove the two maladies to be identical. This is not expected, however, for, as yet, all attempts to transmit corn stripe from corn to cane have given negative results. Cane leaves affected with Cuban streak have also been compared with fixed specimens of the African Streak disease preserved in jars in the laboratory of this station. The stripes of these two affections appear to be quite unlike, and it is believed that they result from the attacks of different causal agents. The insect known to transmit the African Streak disease is not known to occur in Cuba at the present time.

The attacks of the Cuban streak so far observed have been very mild, and the infection has not appeared to spread readily to the surrounding cane. Comparatively little is known about the disease, however, and it must be kept under close observation until its economic importance has been determined.

Cuban streak occurs in the form of interrupted stripes on the leaves. These appear to follow the leaf veins and in most cases extend the full length of the leaf. (Fig. 1.) The color of the stripes is a chlorotic white,

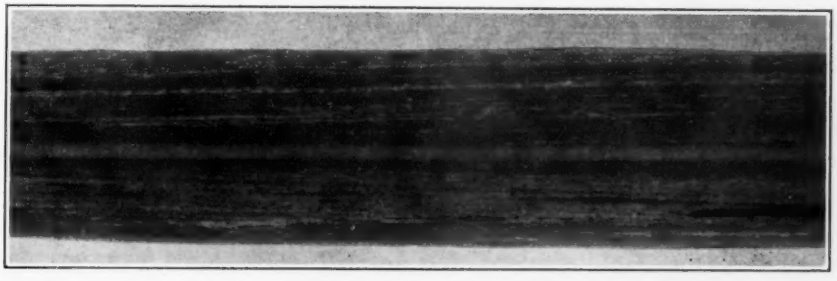

FIG 1. Cuban Streak on POJ 2725 cane. The dark stripes in the picture showed red on the cane leaf.

which shows very plainly against the normal green of the healthy tissues. In some cases some of the stripes change to a brick red, as shown by the dark stripes in figure 1. Nearly all of the leaves on the infected plants usually show from a few to several stripes. The chlorotic white of the stripes shows more distinctly on the dark green of the older leaves than on the lighter green of the younger leaf blades.

The stalks of the infected plants observed appeared to be considerably smaller than the healthy stalks of the same stools, but in most cases they were of about equal height.

The cause of this peculiar striping is not yet known. The appearance of the symptoms, however, strongly suggests the causal agent to be of a virus

nature. This idea is supported by the fact that when the original infected stalk was planted every bud that germinated produced young plants that showed the typical striping on their leaves. These young plants were thickly covered with the stripes until they had grown to a height of about 4 feet. The younger leaves at that time began to show fewer stripes as they came through until the symptoms finally disappeared altogether on the upper leaves of most of the plants. Several insects have been used in preliminary inoculation experiments, but no infection has yet been obtained from them. Since the disease was discovered only a short time ago, it has not been possible to carry out any extensive experiments to determine its cause. Further investigations are necessary before any definite information can be given concerning the nature of the disease or concerning its economic importance.—C. N. PRIODE, Cuba Sugar Club Experimental Station, Central Baraguá, Cuba.

Fusion of Large-cell Hyphae of the Cotton-root-rot Fungus.—In studying the morphology of the cotton root-rot fungus, *Phymatotrichum omnivorum* (Shear) Duggar, during the past season, the writers have observed in artificial cultures numerous cases of specialized fusion of cells of the large-cell hyphae. This development was first observed in cultures of the fungus isolated from infected cotton plants obtained from fields at the U. S. Cotton Breeding Field Station, Greenville, Texas, during the season of 1931. All other isolations of the fungus made from infected cotton plants collected from widely separated localities in Texas have also shown different stages of fusion of the large-cell hyphae. This form of growth occurs to a greater extent in fresh isolations of the fungus, but seems to be entirely absent in attenuated cultures that do not form strands.

It was noted that individual cells of filaments in parallel positions often send out short projections that meet and fuse. In some cases the points of fusion were between the septa, while in others, they were opposite or near the septa; the connecting projections resembling in many respects the conjugations of certain filamentous algae. A simultaneous triple fusion of an individual cell with 2 parallel cells has been noted, after 24 hours incubation in a hanging-drop culture. (Fig. 1.) Other cases have been observed where the cells or filaments grew for several millimeters before encountering and making contact fusions with similar cells of other filaments. Usually at the time of fusion of these hyphae, the protoplasmic contents are multinucleate and appear as hyaline, granular, oval bodies, which refract light readily and give the cells a vacuolate appearance.

Although no actual nuclear fusion of the cells has yet been noted, it is possible that cytological studies may ultimately reveal such phenomena, but there is yet no indication of a reproductive function. The fusion processes appear to represent early stages in the development of the

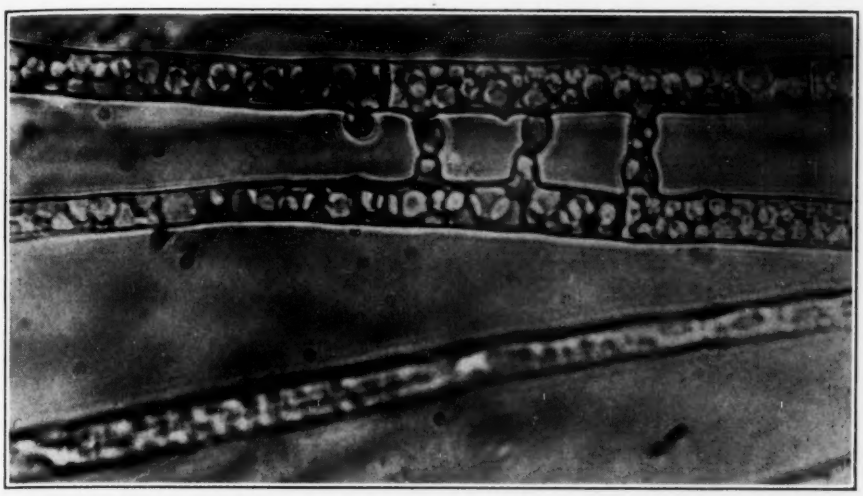

FIG. 1. Photomicrograph of large-cell hyphae of *Phymatotrichum omnivorum* showing simultaneous triple fusion of an individual cell with two parallel cells. ×570.

strand hyphae of the fungus, preceding the development of sclerotia.—D. C. NEAL, R. E. WESTER, and K. C. GUNN, Bureau of Plant Industry, U. S. Department of Agriculture, Greenville, Texas.